Risk Factors for Achilles Tendinopathy in runners – an investigation of selected intrinsic, kinematic, kinetic and muscle activity factors that are associated with Achilles Tendinopathy

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ABSTRACT

The overarching purpose of this study was to investigate the multifactorial aetiology of Achilles tendinopathy. Variables such as training and injury history, lower limb alignment, running biomechanics and movement variability were investigated. This study also aims to understand how different sensory inputs, such as shoe or pain may affect biomechanics. Thirty four uninjured runners (UN) and twenty one runners with Achilles tendinopathy (AT) composed the population sample for this study. Questionnaire and lower limb measurements were used to investigate the multifactorial aetiology of the injury. Selected kinetic, kinematic and muscle activity parameters were employed to analyse the biomechanics aetiology of the injury. Runners performed 10 running trials at a self-selected speed in two running shoes with different hardness. Additionally, the UN runners ran for 10 min on the treadmill while the AT runners ran on the treadmill until they developed pain in the Achilles tendon. The results of the study showed that AT runners have a higher incidence of previous injury, run for more years, have reduced hamstring flexibility, and are heavier and taller than uninjured runners. The study also showed a reduced integrated electromyography activity (IEMG) of tibialis anterior and rectus femoris in the AT group during the running cycle. Stride to stride variability was similar between UN and AT runners but the biomechanics variability between participants were lower for the AT runners. No specific biomechanical adaptations were found between the two different shoe conditions (soft vs. hard). Similarly, biomechanics parameters were not altered at the onset of pain, but the reduced IEMG activity of tibialis anterior and rectus femoris were presented before and during the pain condition. It can, therefore, be concluded that runners with Achilles tendinopathy present reduced muscle activity during running which may be either a novel aetiological factor, or an adaptive response to the injury. The lower variability between runners with AT may indicate that these runners are less able to adjust their biomechanics according to their different functional behaviour or external input signals but this may require further investigation. Finally, it can also be concluded that the sensory inputs such as shoes and pain do not change this muscle activity pattern.

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CHAPTER 1: INTRODUCTION AND SCOPE OF THE THESIS

Achilles tendinopathy afflicts 4.7% of runners (Taunton et al., 2002). Although this is not the most frequent type of running injury, it may be considered unique as it is associated with degenerative changes in the connective tissue structure and a possible underlying genetic basis to its aetiology (Mokone et al., 2006; September et al., 2008). The aetiology of running injuries has been studied for several years, but there is still some controversy about the risk factors associated with running injuries and in particular with Achilles tendinopathy (van Mechelen, 1992; van Mechelen, 1995; Yeung and Yeung, 2001a; Hreljac, 2005; van Gent et al., 2007). Evidence for specific risk factors associated with Achilles tendinopathy is largely limited to descriptive studies relating to clinical experience (McKenzie et al., 1985; Kvist, 1994; Paavola et al., 2002), studies with no control population (Clement et al., 1984) and case control studies (McCrory et al., 1999).

Risk factors for Achilles tendinopathy can be classified into extrinsic and intrinsic risk factors. Intrinsic risk factors are biological and psychosocial characteristics that predispose the runner to injury, while extrinsic risk factors are independent of the runner and associated to the activity during the incident of injury (Taimela et al., 1990). Training errors, footwear and running surface have been suggested as specific extrinsic factors associated with Achilles tendinopathy injury (Smart et al., 1980; Clement et al., 1984; McKenzie et al., 1985; McCrory et al., 1999). Intrinsic risk factors for Achilles tendinopathy are listed as age, gender, muscle weakness, inflexibility, lower limb alignment abnormalities, genetic predisposition and altered biomechanics (kinetics and kinematics) (Kannus et al., 1989; Alfredson et al., 1998b; McCrory et al., 1999; Alfredson and Lorentzon, 2000; Mokone et al., 2006). Traditionally, altered lower limb biomechanical variables refer to changes in kinematics (description of motion) or kinetics (forces that causes or tend to cause change of motion) (Subotnick, 1985; Messier and Pittala, 1988). More recently, altered muscle activity as an additional quantifiable biomechanical risk factor for running injuries has received some attention (Nigg, 2001; Nigg and Wakeling, 2001).

Although running kinematics have been suggested as one of the group of intrinsic risk factors for Achilles tendinopathy, these have not been well studied (McCrory et al., 1999). The most frequently mentioned intrinsic kinematic abnormality associated with Achilles tendinopathy is overpronation of the foot (Clement et al., 1984; Kvist, 1994). Pronation is a triplanar motion which refers to a combination of abduction, dorsiflexion and eversion (Dugan and Bhat, 2005). However, in most of these studies only static lower limb anatomical measurements were used to quantify overpronation (Clancy, Jr. et al., 1976; Clement et al., 1984; Kvist, 1994; Jones, 1998; Paavola et al., 2002). In only one study, two dimensional video data, calcaneal eversion were used to quantify pronation in runners with Achilles tendinopathy (McCrory et al., 1999).

Kinetic risk factors for Achilles tendinopathy have only been reported in one study (McCrory et al., 1999), and there are to our knowledge no studies that have investigated the altered muscle activity as a possible intrinsic risk factor that may be associated with Achilles tendinopathy. The concept that altered neuromuscular control or more specifically muscle activity, may be important in running injury biomechanics was first suggested in 2001 (Nigg and Wakeling, 2001). It was suggested that mechanoreceptors in the sole of the foot sense the impact force during running and that this information is then transmitted to the central nervous system, which then initiates a movement adjustment. According to this hypothesis, the body tries to minimize muscle vibration and, therefore, the muscles are pre-tuned before heel strike.

Recently, it has been suggested that neuromuscular control, resulting in stride to stride variation in lower limb biomechanics (kinematic and kinetic variability), may be an important mechanism to reduce mechanical load to tissues and thereby prevent running injuries (Hamill et al., 1999; Kurz and Stergiou, 2003; Kurz et al., 2003). Variability in any biomechanical parameter can be either within the same participant (intra-participant variability) from stride-to-stride or between groups of participants such as injured compared with uninjured runners (inter-participant variability).

It has been suggested that intra-participant (stride-to-stride) variability in kinematic and kinetic parameters may assist in the reduction of joint loading by a broader distribution of forces in different tissues. This area has not been studied well, but recently it has been reported that that runners with patellofemoral pain display a decreased kinematic variability compared with uninjured runners (Hamill et al., 1999; Heiderscheit et al., 2002). Although intra-participant (stride-to-stride) variability has been studied in runners with patellofemoral pain, there are no studies that have investigated intra-participant variability in runners with Achilles tendinopathy. Furthermore, inter-participant (between-participant) variability in a population of injured runners has not been yet studied.

It is also important to consider the possibility that extrinsic factors such as running shoes may alter intrinsic factors such as kinematics and kinetics. It is frequently suggested that wearing an "incorrect" running shoe is an extrinsic factor associated with a running injury such as Achilles tendinopathy (Smart et al., 1980; Clement et al., 1984; McKenzie et al., 1985). It has been suggested that running shoes reduce impact forces and can control overpronation, and that this will reduce the injury risk (James et al., 1978; Viitasalo and Kvist, 1983; Clarke et al., 1983b; Frederick, 1986). Several studies have investigated the effect of midsole hardness of a running shoe on impact force during running. It has been shown that midsole hardness does not affect vertical impact force peak (Clarke et al., 1983a; Snel et al., 1985; Nigg et al., 1987; Hardin et al., 2004), but may affect vertical loading rate and kinematics (Nigg et al., 1987; Hardin et al., 2004). However, these studies were only conducted in uninjured participants. Possible differences in the kinetic and kinematic responses by wearing shoes with different midsole hardness in an injured population have not been yet studied.

It has also been suggested that extrinsic factors such as shoes, inserts and orthotics may affect muscle activity during running. The effect of footwear (Wakeling et al., 2002b; Nigg et al., 2003; O'Connor and Hamill, 2004; Nigg et al., 2006a) and orthotics (Mundermann et al., 2003; Mundermann et al., 2006) on muscle activity during running has been studied in uninjured runners (Guettler et al., 2006; Kerr et al., 2008). However,

as far as is known, there are no studies that have investigated these responses in an injured population and more specifically in runners with Achilles tendinopathy.

Finally, it important to consider how other sensory inputs may also influence muscle activity during running. Although the mechanism of pain in Achilles tendinopathy is not clearly established (Alfredson et al., 1999; Khan et al., 2000; Alfredson et al., 2001), it is known that runners with mild to moderate (Grade I and II) Achilles tendinopathy classically have no pain at the start a running session. However, pain gradually develops during the running session. It is conceivable that as pain develops, running biomechanics, in particular, muscle activity may be altered. In case-control studies on injured runners, biomechanical measurements are usually only measured during a short period of time during running trials in the laboratory (MacIntyre and Robertson, 1992; McCrory et al., 1999). Therefore, the possible effect of pain, as this develops during running on changes in running biomechanics requires investigation.

Therefore, the overall purpose of this thesis is to explore risk factors that may be associated with Achilles tendinopathy in distance runners. The first question that will be studied is whether there is an association between extrinsic (training history) and intrinsic lower limb alignment factors and Achilles Tendinopathy in runners. This investigation will be followed by studying the possible association between kinetic, kinematic and muscle activity parameters and Achilles tendinopathy in runners. Furthermore, intra-participant and inter-participant variability in kinematic, kinetic, and muscle activity parameters as a possible novel intrinsic risk factor associated with Achilles tendinopathy in runners will be explored. Finally, two further studies will explore how alterations in two external sensory inputs (variations in midsole hardness of the running shoe and the development of pain during running) can affect kinematic, kinetic and muscle activity parameters in runners with Achilles tendinopathy and uninjured runners.

This thesis is therefore divided into a number of Chapters. Following this introductory Chapter, the focus in Chapter 2 is to provide a comprehensive overview of the literature pertaining to lower limb biomechanics and risk factors for running injuries in particular

risk factors for Achilles tendinopathy. Importantly, an evidence-based approach will be followed in the analysis of risk factors for Achilles tendinopathy.

In Chapter 3, research methodology, the description of the participants and the general experimental procedures that was followed by the different studies will be reviewed. The experimental procedures which are specific to each chapter will be further explored further in each pertinent chapter. This chapter also include details of a parallel study that was used to develop a strategy to assist with the decision-making on the electromyography (EMG) normalization method.

In Chapter 4, the relationship between training and lower limb alignment parameters in runners with Achilles tendinopathy will be explored. In this study questionnaire data were obtained, and anthropometrical as well as lower limb alignment measurements were used to explore the relationship between Achilles tendinopathy in runners and possible training factors, anatomical variables and functional characteristics. In Chapter 5, the relationship between Achilles tendinopathy and lower limb kinetic, kinematic and muscle activity variables will be explored. In Chapter 6, stride-to-stride variability (intra-participant) or between participants variability (inter-participant variability) for kinetic, kinematic and muscle activity parameters during running in uninjured runners and runners with Achilles tendinopathy will be investigated. In Chapters 7 and 8, the effects of running shoe hardness and pain that developing during running respectively on kinematic and muscle activity parameters in runners with Achilles tendinopathy will be explored.

In the final chapter (Chapter 9), the findings of the thesis will be summarized and conclusions made in accordance with the purpose and aims of the overall study. Where relevant, recommendations for further studies will be made.

CHAPTER 2: A REVIEW OF LITERATURE

2.1 A Review of the Biomechanics of Running

2.1.1 Introduction

Running has increased in popularity since the 1960's. It is estimated that in the United States of America alone there are over 30 million runners, of which 10 million run on each of more than 100 days per year and around 1 million run to compete in races (O'Connor et al., 2001). The benefits of running include health, fitness, pleasure, relaxation, competition, stress reduction and improvement in personal performance (Clough et al., 1989; O'Connor et al., 2001)

However, a number of epidemiological review studies that investigated more than 500 runners have shown that the annual incidence of running injuries can be as high as 37% - 56% (Marti et al., 1988; Walter et al., 1989; van Mechelen, 1992). The aetiology of running injuries has been associated with a number of postulated extrinsic and intrinsic risk factors (Hess et al., 1989; Rolf, 1995; Krivickas, 1997; Neely, 1998), including biomechanical parameters (Subotnick, 1985; McClay, 2000; DeLeo et al., 2004; Hreljac, 2005; Dugan and Bhat, 2005). Therefore, a better understanding of the biomechanics of running is important to understand the possible mechanism that may lead the development of a running injury. For the purposes of this Chapter, and this thesis, the analysis of running biomechanics will be subdivided into temporal distance parameters and lower limb kinetic, kinematic and muscle activity parameters during running.

2.1.2 Temporal Distance Parameters

2.1.2.1 Introduction

Temporal distance parameters are the first level of analysis in running biomechanics and it involves the measurements of the timing and distance over the running cycle. The

conventional functional unit in gait analysis is the gait cyc mle. One gait cycle is the time between the onset of contact of one foot on the ground to the onset of contact of the same foot. The cycle time (time of a stride length) may vary from about 1 second for walking to less than 0.6 seconds for sprinting (Mann and Hagy, 1980). An increase in running cycle time at similar speed has been associated with increase in energy consumption (Williams and Cavanagh, 1987).

2.1.2.2. Gait cycle and factors affecting the phases of the gait cycle

The gait cycle is further subdivided into a stance and a swing phase. The stance phase of the gait cycle starts at initial contact and finishes at toe-off. Toe-off marks the beginning of the swing phase, which finishes at heel strike (Figure 2.1.). In running, the time of the stance phase is < 50% of the gait cycle, furthermore there are no double stance phases (both feet making contact with the ground) as in walking but there is a period of double float phase (both feet off the ground) (Mann and Hagy, 1980).

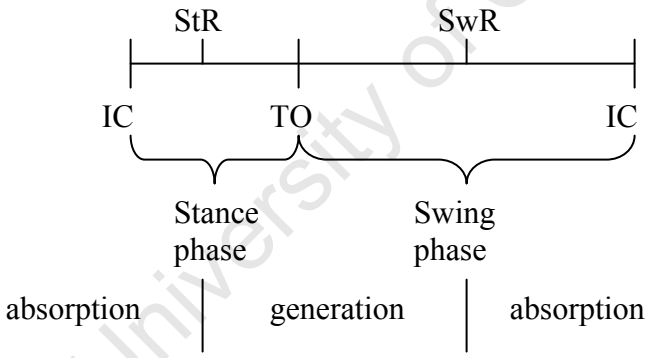

Figure 2.1. The running gait cycle.

IC- initial contact; TO- toe off; StR- Stance phase reversal; SwR- swing phase reversal. Adapted from Novacheck TF., 1998 The biomechanics of running. Gait Posture 7: 77-95.

During the gait cycle, there are exchange periods of acceleration and deceleration, which are referred as absorption and generation. However, onset and completion of these periods do not coincide with the onset of the contact and toe off phases. The absorption period starts at the time of the double float phase, where the centre of mass falls from its peak height and decelerates until stance phase reversal. The generation phase starts at the

time of stance phase reversal, when the centre of mass is propelled upward and forward until the swing phase reversal, where the next period of absorption begins (Figure 2.1.).

During the absorption period of the stance phase, the knee and ankle joints of the supporting limb are flexing, while the joints of the supporting limb are extending during the generation phase. As the running speed increases, the absorption phase decreases and the generation phase increases (Ounpuu, 1990), (Figure 2.1).

An increase in running speed is initially generated by an increase in stride length. However, once optimal stride length is achieved, further increases in running speed can only occur with an increase in stride frequency. Stride length is limited by individual height, limb length and running ability (Birrer et al., 2001). There is evidence that runners naturally select a stride length that is more economical according to their individual characteristics. In a study of 45 recreational runners, it was shown that 80% of runners naturally selected a stride length that was within 5% of their optimal economical stride length (Morgan et al., 1994).

Other than the intrinsic factors there are extrinsic factors, which can affect stride length. Extrinsic factors that can affect stride length are sloping surface (Paradisis and Cooke, 2001), treadmill compared with over ground running (Wank et al., 1998) and the type of running surfaces (Pinnington et al., 2005). It has been documented that running on a treadmill is associated with a shorter stride length, increased stride frequency, and shorter contact time when compared with running on the ground (Wank et al., 1998).

In an attempt to relate ground reaction force with temporal distance parameters (Mercer et al., 2002), it has been found that the shock attenuation during running is highly correlated with stride length (r=0.71) but only moderately correlated with stride frequency (r=0.40). Shock attenuation is defined as the process to reduce the magnitude of the impact between the leg and the head (Mercer et al., 2002). It has also been shown that shock attenuation tends to increase with the increase in stride length with a concurrent decrease in stride frequency (Derrick et al., 1998).

2.1.2.3 Summary

It can be concluded that temporal distance parameters are affected by intrinsic factors (e.g.: height, limb length and running ability) and extrinsic factors (slope and type of surface, treadmill). Moreover, the increase in stride length is associated with the increase in shock attenuation.

2.1.3 Kinetics of Running

2.1.3.1 Introduction

Kinetics is defined as the study of forces that cause movement. The kinetics of running have been well investigated in relation to running speeds (Queen et al., 2006), footwear (Clarke et al., 1983a), injuries (Zifchock et al., 2006), running economy (Martin et al., 1993), running techniques (Arendse et al., 2004), slope (Gottschall and Kram, 2005) and hardness of surfaces (Dixon et al., 2000). The reaction force from the ground provided during movement is known as ground reaction force (GRF) (Hamill and Knutzen, 2003) and it has three components: anterior-posterior, medial-lateral and vertical.

2.1.3.2 Anterior-posterior forces

The anterior-posterior forces have two phases: braking and propulsive (Ounpuu, 1990). The anterior-posterior force presents a negative component in the first half of the contact phase due to horizontal friction between the shoe and the surface; it is termed horizontal braking force (HBF). In the second half of the contact phase, the anterior-posterior force is known as the horizontal propulsive force and the positive shift reflects the force generation of the foot pushing back on the ground (Figure 2.2).

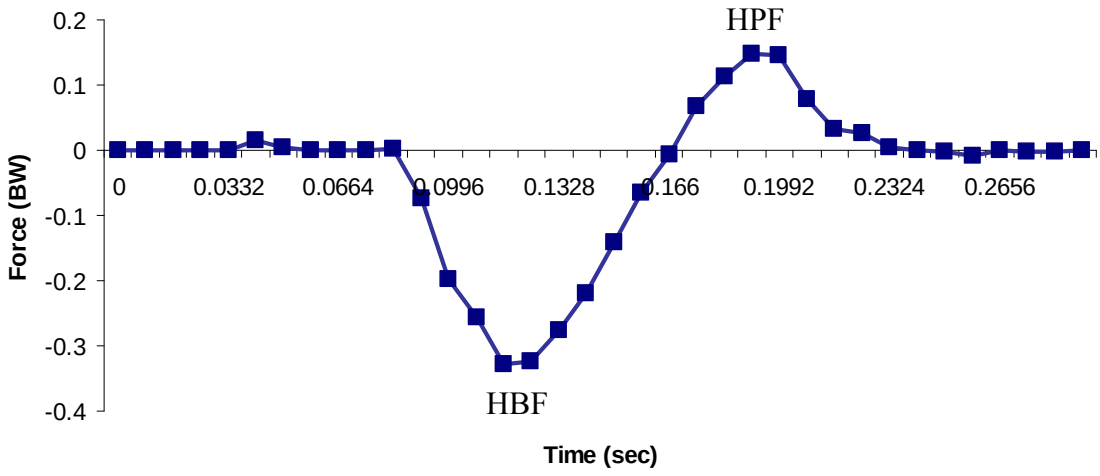

Figure 2.2. Anterior-posterior ground reaction force running (HBF-horizontal braking force; HPF-horizontal propulsive force)

2.1.3.3. Medial-Lateral Forces

Medial–lateral force is the smallest of the three components of the GRF in linear running. The magnitude of medial-lateral force ranges from 0.01 body weight (BW) to 0.1 BW and the pattern is variable according to the position of the forefoot adduction and abduction (Simpson and Jiang, 1999). It has been stated that there is a large inherent variability of the medial-lateral GRF between participants, which limits the use and interpretation of this variable (Munro et al., 1987). Therefore, medial-lateral GRF will be not be discussed further or analysed in this thesis.

2.1.3.4. Vertical impact forces

The magnitude of the vertical impact force during walking can reach 1.3 to 1.5 times the body weight during loading and push–off. In running, the magnitude of the vertical impact force is usually around 2 to 3 times the body weight, but it increases with increasing running speed (Munro et al., 1987).

The GRF displays a specific pattern during running which has been well-described and has two distinct peaks. The first peak occurs just after initial contact and it is called vertical impact force peak (VIF) or passive peak. This use of the terminology "passive" relates to the fact that this phase is not under muscular control (Hamill and Knutzen, 2003). The magnitude of the first peak is influenced by running speed, lower limb joint angles, surface stiffness and the area of contact between the foot and the surface (Dixon et al., 2000). The second GRF peak is usually of a higher magnitude and is referred as vertical propulsive force peak (VPF) or active peak. The terminology "active" refers to the role that muscle activity plays in development of this force (Figure 2.3) (Hamill and Knutzen, 2003).

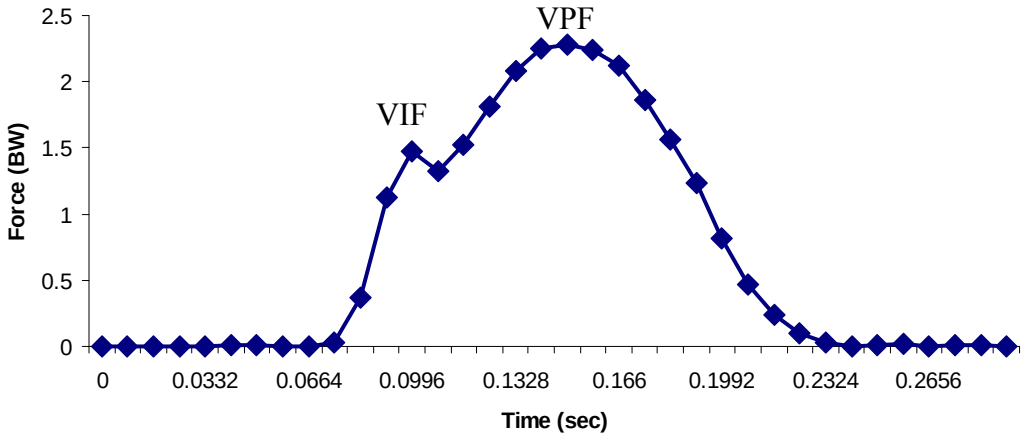

Figure 2.3. Vertical ground reaction force during running (VIF-vertical impact force peak; VPF- vertical propulsive force peak)

2.1.3.4.1 Vertical Loading rate

Apart from the measurement of the peak force, a second important measurement in vertical GRF is the loading rate. Vertical loading rate is defined as the change in force related to time (Nigg et al., 2000). It can be described as the slope of the force-time curve. Recently, increased loading rates during running have been associated with the development of running injuries (Hreljac et al., 2000; Gerlach et al., 2005). There is also a strong correlation between loading rate and vertical impact force peak (r=0.98) (Frederick, 1984). It has been suggested that loading rate is a more important

biomechanical variable to analyse than vertical impact force peak (Munro et al., 1987). The argument for this is that forefoot strikers do not display a visible impact force peak (Figure 2.3). There is some variation in the calculation of the loading rate. Loading rate has been calculated by determining the time for the vertical force to rise from 50N to body weight (BW) plus 50N (Munro et al., 1987). However, this method has been criticized by some authors (Miller, 1990), because the selection of 1 BW is arbitrary and other forces ranges could be equally applied. More recently, vertical loading rate (VLR) has been defined as the VIF divided by the time to reach the VIF (Hargrave et al. 2003) and this is the preferred method that will be used to define VLR in this thesis.

2.1.3.5 Factors affecting ground reaction forces

It has been shown that running speed can affect the magnitude, but not the timing, of ground reaction force (Ounpuu, 1990). Additionally, the magnitude of the GRF increases linearly with increased speed and is linearly correlated with loading rate (Keller et al., 1996).

According to results from recent studies, impact force peak and maximal loading rate are higher in older (55-65 years) compared with younger (20-35 years) runners at a controlled speed (Bus, 2003). It has been speculated that this change in shock-absorbing capacity in older runners may be related to increased injury risk. Equal GRF has been reported in male and female participants during walking and running at similar speed (Keller et al., 1996). It has also been shown that more economical runners have lower vertical impact force peak and antero-posterior peak forces at the same running speed (Williams and Cavanagh, 1987).

It has also been suggested that the running surface can affect GRF. Although it is expected that increase in surface hardness would increase GRF, recent studies have shown that vertical forces peaks are maintained when running on surfaces of differing hardness (Ferris et al., 1999; Dixon et al., 2000). The similar GRF, which is observed when running on surfaces of differing hardness, is probably related to lower limb

kinematic adaptations. It is interesting to note that although vertical impact force peak is not altered between different surfaces, the vertical loading rate may vary (Dixon et al., 2000). The association between an increase in surface hardness and an increase vertical loading rate may indicate that this variable is a better indicator of cushioning ability than peak impact force which was unaltered according to different surfaces.

The slope of a running surface can also affect GRF. In a recent study, it has been shown that a negative slope of 9° (downhill running) can increase impact force by 54% compared with level running (Gottschall and Kram, 2005). Walking on a treadmill has been shown to promote lower peak values of GRF when compared with walking on overground surface (Riley et al., 2007). However, there appear to be no comparable studies of GRF on overground and treadmill running.

Running technique is another mechanism that can alter GRF. An increase in knee flexion at initial contact (Derrick, 2004), together with an increase in ankle dorsiflexion and pronation (Hintermann and Nigg, 1998), have been shown to be an important mechanisms to reduce impact force. A reduction in impact force due to kinematic alterations was observed in a study that investigated a novel running style (Pose method) (Arendse et al., 2004). The mechanism of reducing vertical impact force and loading rate was associated with a higher knee flexion at initial contact running, and consequently a reduction in the eccentric load on the knee compared with heel-strike running technique.

Knee flexion excursion, which can be defined as the difference between knee angle at peak vertical force and initial contact, has also been inversely related to peak vertical forces and loading rate on landing (Hargrave et al., 2003). In a mathematical model, which describes the dissipation of impact force at heel strike, it was estimated that 70-80% of impact reduction could be attributed to the knee joint (Kim et al., 1994). However, knee flexion is thought to occur together with foot pronation to reduce impact force more effectively (Hamill et al., 1992).

Fatigue that is induced by maximal exercise on a treadmill can also reduce vertical impact force peak and loading rate by 6% and 11% respectively (Gerlach et al., 2005). In This study the reduction in GRF noted during the fatigue exercise was associated with an observed decrease in stride frequency and an increase in step length and maybe some speculative changes in lower limb kinematics.

Finally, footwear (Frederick, 1986) and orthotics (Mundermann et al., 2003) can also influence GRF and will be explored further in section 2.4 *A review of the biomechanics of running shoes.*

2.1.4 Kinematics of Running

2.1.4.1 Introduction

Kinetics refers to the description of motion, which include the pattern and speed of movement (Hall, 2007). The kinematics of running is conventionally discussed in the three planes of motion: sagittal, frontal and transversal.

2.1.4.2 Sagittal plane kinematics

The sagittal plane movement is the plane of motion that most contributes to the kinematic changes that are observed in walking and running, as this is the most effective plane of motion to increase velocity in the direction of progression. The kinematic differences that are observed in the frontal and the transverse plane become relatively smaller as running speed increases (Ounpuu, 1990).

During running, the hip is flexed at initial contact (around 45 degrees) and then continually extends (5 degrees) to toe-off (Ounpuu, 1990). Maximum hip extension occurs just before toe-off and maximum hip flexion occurs between mid and terminal swing phase (approximately 45 degrees). As running speed increases, the hip extension

angle remains the same but hip flexion increases to promote a longer step length (Novacheck, 1998b).

During the absorption period, the knee flexes to approximately 45 degrees at midstance and is followed by knee extension of 25 degrees by the end of stance phase. During the swing phase, the knee flexion stays around 90 degrees. With an increase in running speed, there is a reduction of knee flexion during the initial absorption period in the stance phase and there is an increase in knee flexion in the swing phase (Mann and Hagy, 1980).

The initial contact of the foot on the ground normally occurs on the heel for walking and running. It is estimated that 80% of distance runners are rearfoot (heel) strikers and the remainder are considered mid foot strikers (McClay, 2000). As the body moves from initial contact to mid-stance phase, there is an increase in ankle dorsiflexion. During the mid to terminal stance phase the ankle moves to plantar flexion and in the swing phase the ankle position moves to dorsiflexion. As the running speed increases, the initial contact tends to occur on the forefoot, there is a reduction in dorsiflexion in the absorption period in the stance phase and there is a decrease in dorsiflexion in the swing phase (Novacheck, 1998b). The movement of the ankle joint is considerably different between walking and running. During walking there is a plantarflexion at initial contact followed by dorsiflexion, whereas during running there is an opposite motion - the initial contact occurs in dorsiflexion and then progresses to plantarflexion (Mann and Hagy, 1980).

In relation to the range of motion (ROM), there is an increase of hip, knee and ankle ROM with an increase in running speed (Mann and Hagy, 1980). The increase in the ROM of the hip and knee occurs mainly due to an increase in flexion, whereas joint extension tends to decrease as the running speed increases.

2.1.4.2.1. Factors affecting sagittal plane kinematics

A number of factors have been shown to affect sagittal plane kinematics during running. These include gender, age, surface hardness and running on a treadmill.

A study comparing knee kinematics has found that female runners have less peak knee flexion and knee excursion than male runners during running (Malinzak et al., 2001). However, this is not a consistent observation and similar sagittal plane kinematics between gender were reported in another study (Ferber et al., 2003).

The possible effect of age on sagittal plane kinematic parameters during running has also been studied (Bus, 2003). It has been have found that older runners had an increase in knee flexion at heel strike, and decreased ROM in the stance phase during running compared with younger runners.

As previously mentioned, in studies comparing different running surfaces, peak impact force was consistent across surfaces with different hardness. However, kinematic adaptations such as 1) an increase in knee flexion at initial contact (Dixon et al., 2000), and 2) a decrease in leg stiffness (Ferris et al., 1999) (as measured by the ratio of peak vertical ground reaction force and vertical displacement of the centre of mass) were observed when running on harder compared with softer running surfaces.

Kinematic changes were also reported in a study investigating the effects of running surfaces with different gradients (Paradisis and Cooke, 2001). In this study, it was shown that, at initial contact during downhill running, there was a significant reduction in hip flexion and an increase in knee extension when compared with running on a level surface. In the same study, it was also found that uphill running was associated with a decrease in hip angle and shank angle at toe-off, compared with running on a level gradient.

The effects of overground compared with treadmill running on sagittal kinematic parameters have also been investigated (Nigg et al., 1995; Wank et al., 1998). The results of these studies indicated that runners tend to land with the foot in a more neutral position when running on a treadmill compared with overground running. Furthermore, there was also a trend for runners to have a reduced knee angle at initial contact and reduced hip ROM in treadmill compared with overground running (Wank et al., 1998). However, in these studies there was also a large inter-participant variability in these kinematic variables. Based on these studies it has been concluded that biomechanical assessment during treadmill running can lead to different findings when compared with overground running (Nigg et al., 1995; Wank et al., 1998).

These studies, therefore suggest some intrinsic (e.g. age and gender) and extrinsic (e.g. running surface) may affect sagittal plane kinematics during running and these parameters therefore have to be taken in consideration during the design of a study.

2.1.4.3 Frontal plane kinematics

In the frontal plane, the motion of the knee and ankle joints is minimal because these are restrained by the collateral ligaments of the knee and ankle (Novacheck, 1995; Novacheck, 1998b). However, movement of the hip in the frontal plane during running is important in order to minimize upper body movement during running (Novacheck, 1998b). At initial heel contact during running, the hip adducts relative to the pelvis (approximately 6 degrees). This frontal plane movement is an important shock absorbing mechanism. The hip then remains adducted as the limb is loaded and moves to abduction from the mid-stance to the mid-swing (6 degrees) phase of running. The hip then returns to adduction during the late-swing phase (Ounpuu, 1990).

2.1.4.3.1. Factors affecting frontal plane kinematics

There are very few studies that have examined the possible factors that may affect frontal plane kinematics during running. It does appear that gender may affect frontal plane

kinematics during running as it has been shown that female runners have greater hip adduction and knee abduction throughout the stance phase of running (Malinzak et al., 2001; Ferber et al., 2003).

2.1.4.4 Transverse plane

Transverse plane kinematics of the pelvis and foot during running has been studied. It has been observed that during running, the pelvis is externally rotated at initial heel contact (8°), and then moves to the neutral position at mid-stance followed by internal rotation at toe-off (8°) (Ounpuu, 1990). Therefore, the hip internally rotates at heel strike to mid-stance and then externally rotates throughout the stance phase until toe-off (Ferber et al., 2003).

Foot kinematics in the transverse plane during running has also been studied. It has been shown that the foot externally rotates from initial contact to mid-stance and then internally rotates to neutral to toe-off (Ounpuu, 1990). Additionally, the movement coupling between the foot and the shank during initial contact to midstance results in the tibia to be rotated internally (Stacoff et al., 2000). However, the magnitude of motion in the transverse plane during running is small when compared with the motion observed in the sagittal plane (Novacheck, 1998b).

2.1.4.4.1. Factors affecting transverse plane kinematics

Factors affecting transverse plane kinematics during running have not been studied extensively. In one study, it has been shown that female runners appear to have increased hip internal and knee external rotation during the stance phase when compared with male runners (Ferber et al., 2003).

2.1.4.5 Subtalar joint pronation and supination

Over the past 3 to 4 decades, there has been considerable interest in the movements of pronation and supination of the foot and ankle. Pronation in particular have been linked to injury (Stacoff et al., 1991; Stergiou et al., 1999; Hreljac et al., 2000) and this has resulted in the development of changes in the footwear industry. Therefore, a discussion of these movements during the gait cycle, as well as factors affecting these movements, is appropriate.

It is important to point out that pronation and supination is a triplanar motion of the subtalar joint. Subtalar joint pronation comprises a combination of abduction, dorsiflexion and eversion, which are derived from the transverse, sagittal and frontal plane respectively. Subtalar joint supination, on the other hand, includes the motion of adduction, plantarflexion and inversion (Dugan and Bhat, 2005). Pronation and supination are important in assisting movement, stabilizing the joints and reducing impact forces of the lower limb (Donatelli, 1985).

Subtalar joint pronation occurs during the absorption phase of the stance and provides shock absorption while supination occurs during the generation phase of the stance phase and provides a stable lever for push-off (Novacheck, 1998b). More specifically, at terminal swing phase and heel strike the foot is in a supinated position. From heel strike to 20% of the stance phase, the foot moves from supination to slightly pronation. Pronation is then maintained from 55-85% of the stance phase. Maximum pronation normally occurs between 35-40% of the stance phase. As the foot moves from the absorption to the generation phase, it returns to neutral position (approximately 70 - 90% of stance) and then to supination at the toe-off (Rodgers, 1995).

The measure of calcaneus eversion has been indistinguishably mentioned in two dimensional kinematics studies as “pronation”. Studies with two dimensional video analysis have reported an average 9.4° of maximum “pronation” and have considered maximum “pronation” values higher than 13° and total rearfoot range of motion higher than 19° to be excessive (Clarke et al., 1984). There is a common belief that runners who overpronate have a higher risk of sustaining injury (James et al., 1978; Smart et al., 1980;

Willems et al., 2007). This is further explored in Section 2.2 (*A Review of the Epidemiology of Running Injuries*). There are several factors that may affect pronation such as: lower limb alignment, flexibility, use of orthotics and running shoes. These also will be further explored later in this chapter (Section 2.2.4 *General risk factors associated with running injuries*).

In summary the description of normal running kinematics can be described in the three planes of movement. However, there are many intrinsic and extrinsic factors which can affect running kinematics and should be taken in consideration when reviewing studies on running kinematics, or when research studies are designed.

2.1.5 Muscle activity during running

2.1.5.1. Introduction

The terms neuromuscular control and muscle activity are often used interchangeably in the literature. However, when electromyography (EMG) activity of the muscles of the lower limb is measured during running the more correct term to describe this parameter is muscle activity rather than neuromuscular control. Therefore, in this thesis, muscle activity will be used consistently where EMG data of muscles during running is described.

An analysis of muscle activity during running is essential to the understanding of the biomechanics of running. Recently, the analysis of muscle activity during running has become important in understanding the biomechanics of running injuries (McClay et al., 1990; Nigg and Wakeling, 2001). Muscle activity during walking and running in uninjured runners has been well reported in the literature (Mann and Hagy, 1980; Ounpuu, 1994; Novacheck, 1998b; Mundermann et al., 2003). However, muscle activity of injured runners during running is an area that still needs further investigation (Weist et al., 2004; Delahunt et al., 2006). In this section, muscle activity during running will be reviewed. In particular, the following three aspects related to muscle activity will be

reviewed: 1) measurement of muscle activity during running, 2) patterns of muscle activity in different lower limb muscle during running, and 3) factors affecting muscle activity during running.

2.1.5.2. Measurement of muscle activity during running

As mentioned electromyography (EMG) is the main measurement technique to document muscle activity during running. The electromyogram using fine needle or surface electrodes is used to measure the electrical activity of the motor units from the muscle (Nigg et al., 2000). When studying muscle activity using EMG, it is important to note that there is an approximate 50 ms delay between the onset of EMG activity and the development of muscle force (Sherif et al., 1983).

EMG activity can be reported using two types of variables: amplitude and frequency (Kamen and Caldwell, 1996; Kamen, 2004). The variables that are used to define EMG amplitude are: 1) peak to peak amplitude, 2) average rectified amplitude, 3) root mean square amplitude, 4) linear envelope, and 4) integrated electromyography (IEMG) (Kamen, 2004). With regard to EMG frequency, the most common methods are turning points or zero crossing and mean or median frequency. Other than amplitude and frequency, onset/offset analysis of EMG activity can also be applied if it is of interest to document the start and the end of muscle activity over a time period, such as the gait cycle (Kamen, 2004).

2.1.5.3. Lower limb muscle activity during running

The patterns of muscle activity of the common larger lower limb muscles during running have been well documented (Mann and Hagy, 1980; McClay et al., 1990; Ounpuu, 1990; Novacheck, 1998b). The muscle activity of these muscles during the gait cycle will now be discussed but are summarised in Figure 2.4.

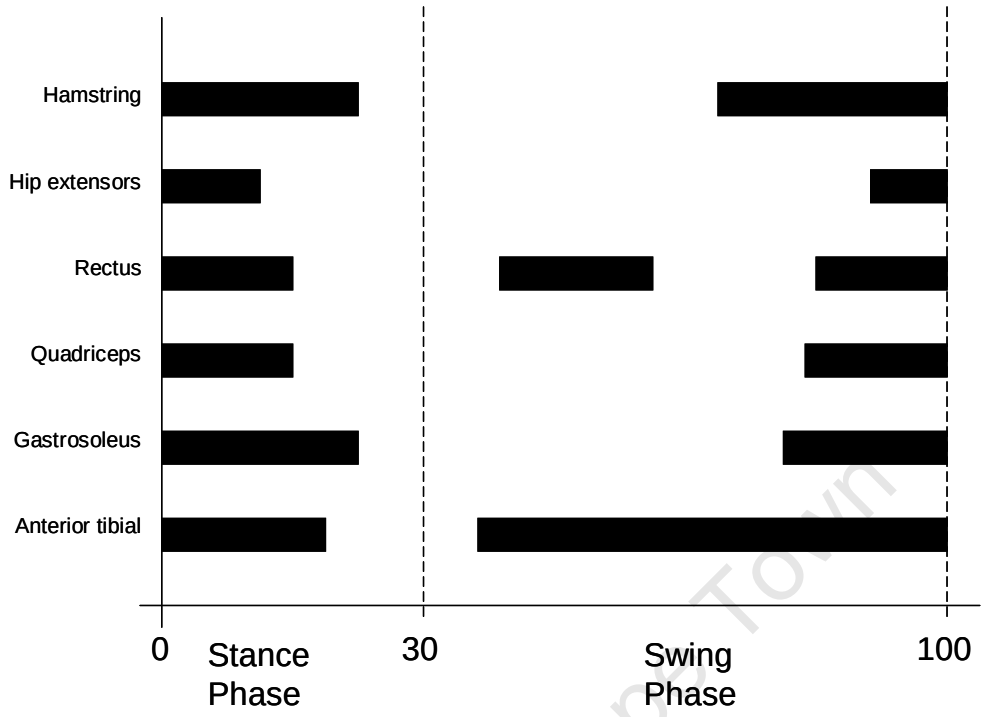

Figure 2.4. Muscle activity during a gait cycle.

Adapted from Novacheck TF., 1998 The biomechanics of running. Gait Posture 7: 77-95.

2.1.5.3.1. Quadriceps muscle activity during running

The quadriceps muscle prepares the lower limb for ground contact and also absorbs impact during the absorption phase of the stance phase. Therefore, peak activity of the quadriceps muscle occurs during the late swing phase (last 20%) and the early stance phase (first 50%) of running (Figure 2.4.). In the early stance phase, the quadriceps muscle contraction is mainly eccentric (i.e. lengthens under tension) and restrains the tibia as the knee flexes (Mann and Hagy, 1980; McClay et al., 1990; Ounpuu, 1990; Novacheck, 1998b).

The onset of rectus femoris activity is around 87% of the gait cycle or approximately 78ms before initial heel contact, and this is related to the development of muscle force just before initial contact (Novacheck, 1998b). The earlier activity of rectus femoris during the swing phase (Figure 2.4) is perhaps related to the hip flexor function of this

muscle (McClay et al., 1990). Unlike the rectus femoris, the vastus lateralis and vastus medialis do not span across the hip joint, therefore they do not assist in hip flexion but may help to control knee flexion during stance phase (McClay et al., 1990).

2.1.5.3.2. Hamstring muscle activity during running

The hamstring muscle group is active at the second half of the swing phase (last 40%) and first half of the stance phase. The hamstring muscles are responsible for decelerating the momentum of the tibia as the knee extends before initial heel contact (Novacheck, 1998b). Additionally, this muscle group co-contracts with the quadriceps to provide stability during heel contact (McClay et al., 1990).

2.1.5.3.3. Tibialis anterior muscle activity during running

The tibialis anterior muscle is active throughout the gait cycle. However, the maximum activity occurs at initial heel contact and during the absorption phase, when it acts eccentrically to control plantar flexion (lowering of the foot on the ground) (McClay et al., 1990). There is also an increase in the tibialis anterior muscle activity in the transition between the absorption and the generation phase, when it acts concentrically to provide foot clearance. There is constant activity of this muscle throughout the swing phase to control plantar flexion and to initiate dorsiflexion (McClay et al., 1990; Ounpuu, 1990; Novacheck, 1998b).

2.1.5.3.4. Gastrocnemius-soleus muscle activity during running

The gastrocnemius-soleus muscle complex is active from late swing phase through 50%-80% of the stance phase (McClay et al., 1990). During the late swing phase and the early stance phase, the gastrocnemius-soleus muscle complex acts eccentrically, and co-contracts with the tibialis anterior to stabilize the foot position for heel strike and to decelerate the movement of the shank. Peak activity of this muscle complex occurs at 25% of the stance phase after which point the muscle activity reduces until toe-off.

During the generation phase, there is concentric muscle contraction of this muscle complex to produce plantar flexion (Ounpuu, 1990).

2.1.5.3.5. Hip abductor muscle activity during running

The hip abductor muscles (tensor fasciae latae, gluteus medius, and gluteus minimus) are active during the late swing and the early stance. These muscles act to prepare the foot for contact and to provide hip stabilization during the early stance phase of running (McClay et al., 1990).

2.1.5.3.6. Peroneus muscle activity during running

The peroneus muscles (peroneus longus and brevis) are responsible for eversion and plantar flexion of the foot. This muscle group provides lateral stability of the foot during the stance phase. The peroneus muscles are active on the early stance phase to increase joint stiffness and to provide stability (O'Connor and Hamill, 2004).

2.1.5.4. Factors affecting lower limb muscle activity during running

In recent years, there has been considerable interest in studying the possible factors that can affect muscle activity during running. Factors that may affect muscle activity during running include the following: shoes with different midsole hardness (Wakeling et al., 2002b; Nigg et al., 2003; Boyer and Nigg, 2004); midsole shape (O'Connor and Hamill, 2004; Nigg et al., 2006a); orthotics (Mundermann et al., 2006); barefoot running (von Tscharner et al., 2003); running speed (Kyrolainen et al., 2005); fatigue (Weist et al., 2004); running surface (Pinnington et al., 2005); and treadmill compared with overground running (Wank et al., 1998). Studies that have investigated the effect of barefoot running or running with shoes with different midsole hardness, shape and orthotics on muscle activity will be explored further in Section 2.4 (*A review of the biomechanics of running shoes*).

2.1.5.4.1. Running speed and muscle activity

Running speed affects lower limb muscle EMG activity (Nilsson et al., 1985; Simonsen et al., 1985; Kyrolainen et al., 2005). An increase in running speed is associated with an increase in the amplitude of the muscle activity of the hip extensors (Nilsson et al., 1985; Simonsen et al., 1985). However, when running speed is increased to a point when fatigue occurred, studies have shown that EMG activity is then reduced (Weist et al., 2004; Nummela et al., 2006).

2.1.5.4.2. Running surfaces and muscle activity

Running on different surfaces affects lower limb muscle activity during running. In one study, it has been shown that EMG amplitude was greater when running on sand compared with running on a firm surface (Pinnington et al., 2005). It was suggested that this increase of EMG activity was related to the higher energy cost of running on sand.

It has also been shown that running on a treadmill appears to increase muscle activity amplitude of the biceps femoris. The explanation for this observation is not clear, but the authors have linked this increased activity to the forward leaning of the trunk that was observed during treadmill running compared with overground running (Wank et al., 1998). In the same study, it was also found that muscle activity of the vastus lateralis was reduced during treadmill running, and this was related to the observed reduction in vertical displacement on the treadmill when compared with overground running (Wank et al., 1998).

2.1.6 The stretch-shortening cycle

It is well known that if the muscle contracts while it is actively stretched (eccentric contraction), it can perform considerably more work during a subsequent concentric contraction compared with a concentric contraction without pre-stretching. This pattern of eccentric followed by concentric contraction is defined as the stretch- shortening cycle

(SSC) (Nigg et al., 2000; Hall, 2007). In addition to the kinetic, kinematics and muscle activity measurements, a study of the SSC in muscle groups during running provides a further dimension to the understanding of the biomechanics of running and deserves brief discussion.

An important contributor in the stretch-shortening cycle is the series elastic component (SEC), which resides in the tendon and acts as a spring to store elastic energy and increase force production (Hall, 2007). It is recognized that tendon elasticity is more important than the muscle elasticity to store elastic energy (Nigg et al., 2000). For a 70kg runner, the kinetic and gravitational energy lost and regained at each heel strike is around 100 J. However, half of this energy is stored as strain and returned as elastic recoil by the Achilles tendon (35J) and the ligaments (17J) while the other half is supplied by muscular work (Ker et al., 1987).

Taking into consideration the importance of the tendon in the SSC, it can be hypothesised that exercise performance may be influenced not only by the force and power provided by the muscles, but also by the viscoelastic properties of the tendon structures. In a recent investigation, concentric torque in a SSC exercise was negatively correlated with muscle tendon stiffness and hysteresis during a ramp isometric plantar flexion exercise (Kubo et al., 2005). Hysteresis can be defined by the amount of energy lost as heat during the recoil from stretch. The same authors concluded that performance in this type of exercise can be negatively affected by the viscoelastic properties of tendon structures (Kubo et al., 2005). Other than the tendon itself, another potential contributor to the SSC is the muscle spindle which initiates the stretch reflex and is stimulated by the force lengthening of the muscle (Trimble et al., 2000).

Muscles, tendons and ligaments perform as a spring during running and as speed increases there is an increase in spring stiffness (Hall, 2007). In one of the earliest studies, EMG activity of the leg extensors and flexors muscles during running has been studied in conjunction with muscle-tendon unit length (Ker et al., 1987). In this study, it was documented that, with the exception of the rectus femoris, which was active only at

late swing phase, all the other muscles studied were active during mid-swing phase, possibly in preparation for the impact forces during landing. It was speculated that the eccentric contraction of the hamstring and gluteus maximus during the swing phase probably results in the storage of elastic energy, which is then released during the support phase of the gait cycle (Ker et al., 1987). Thus, the tendon unit is an important storage of elastic energy during the stretch shortening cycle and the viscoelastic proprieties of the tendon may affect the stretch shortening cycle and possibility the mechanisms of running.

2.1.7 Summary and Conclusion

In this section, the normal biomechanics of running and the associated kinetic, kinematics and muscle activity parameters have been reviewed. Notably, several factors have been shown to affect kinetic, kinematics and muscle activity parameters during running. These factors include the following: age, gender, running speed, fatigue, tendon stiffness, shoes and running surface. It was important to first review normal running biomechanics, and the factors that influence normal biomechanics before study biomechanics of injured runners and factors that may contribute to the development of a running injury.

The focus of this review Chapter will therefore now shift to running injuries. After a brief review of the epidemiology of running injuries, specific risk factors associated with running injuries will be reviewed. Thereafter, the emphasis will shift to Achilles tendinopathy, which is the specific running injury that was studied in this thesis.

2.2 A Review of the Epidemiology of Running Injuries

2.2.1. Introduction

Epidemiology refers to "*the study of the distribution of diseases and determinants of disease in populations*" (Oxford Dictionary, 2007). In epidemiological terms the incidence of a running injury can be defined as the percentage of injuries in a population of runners per annum (annual incidence) or as injuries per hours of running (injuries per

1000 hours running) (Lysholm and Wiklander, 1987; van Mechelen, 1992). It has been reported that the annual incidence of injury in runners varies between 30-65% (Marti et al., 1988; Walter et al., 1989; Macera et al., 1989; van Mechelen, 1992; Hoeberigs, 1992) but can be as high as 90% in marathon runners (Fredericson and Misra, 2007). However, the annual incidence of running injuries is still 2 to 2.5 times less frequent than injuries in other sports (Marti et al., 1988).

As indicated, the incidence of injury can also be expressed as injury per time of exposure (injuries per 1000 hours of training). In this respect it has been shown that the incidence of running injuries in marathon runners (2.5/1000 hours) is lower when compared with middle distance runners (5.8/1000 hours) or sprinters (5.6/1000 hours) (Lysholm and Wiklander, 1987).

Most running injuries are classified as overuse injuries. An overuse injury can be defined as an injury of the musculoskeletal system that results from fatigue of a certain structure of the body that has been stressed over a period of time (Hreljac, 2005). According to the stress-frequency curve (Figure 2.5), the risk of an overuse injury is related to both the magnitude of the stress applied as well as the frequency of application of this stress. In the case of distance running, frequency can be related to the distance travelled, stride frequency (running speed) or even the time period between each run (Hreljac, 2005). It is, however, important to note that high magnitudes of stress on the tissue can result in injury, whereas lower (physiological) stress, usually defined as less than the tissue failure limit, is an important stimulus for remodelling and adaptation of the tissue.

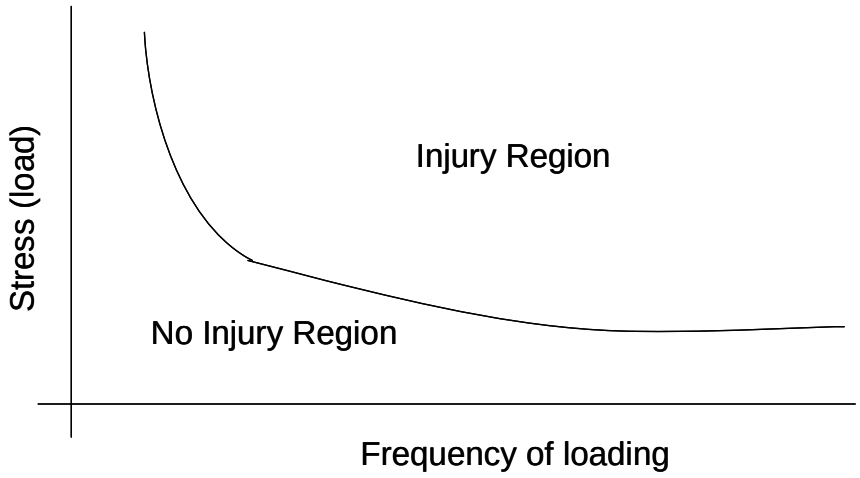

Figure 2.5. Stress-frequency curve.
Adapted from Hreljac, A. (2004). Impact and overuse injuries in runners. Med.Sci.Sports Exerc. 36**,** 845-849.

2.2.2 Distribution of running injuries

The site of running injury may be specified by the anatomical site in which this injury occur (Taunton et al., 2002) or by the type of injury (Clement et al., 1981; Pinshaw et al., 1984; Taunton et al., 2002; van Gent et al., 2007; Fredericson and Misra, 2007).

In a retrospective case-control analysis, it was documented that the knee is the most common anatomical site of injury (42.1%) in runners. This is followed by the foot and ankle (16.9%), lower leg (12.8%), hip/pelvis (10.9%), Achilles tendon/calf (6.4%), upper leg (5.2%), lower back (3.4%) and other anatomical areas (2.2%) (Taunton et al., 2002).

According to several studies, the most common type of injury is the patellofemoral pain syndrome (PFPS), which accounts for 17-50% of running injuries (Clement et al., 1981; Pinshaw et al., 1984; Taunton et al., 2002; van Gent et al., 2007; Fredericson and Misra, 2007). The frequency of other common running injuries, that follow the patellofemoral pain syndrome, differ according results from various studies (James et al., 1978; Clement et al., 1981; McKenzie et al., 1985; Taunton et al., 2003). The frequency of running injuries, from one of the largest and most recent retrospective studies reporting on 2002 running injuries, is depicted in Table 2.1 (Taunton et al., 2002).

Table 2.1 Frequency of the most common running injuries Adapted from: Taunton JE et al., 2002. A retrospective case-control analysis of 2002 running injuries. Br J Sports Med 36: 95-101.

Injury	Total frequency (%)
Patellofemoral pain syndrome	16.5
Iliotibial band friction Syndrome	8.4
Plantar Facsiitis	7.9
Meniscal injuries	5.0
Tibial stress syndrome	5.0
Patellar Tendinitis	4.7
Achilles Tendinitis	4.7
Gluteus medius injuries	3.4
Others	44.4

As the focus of this thesis is on one of these running injuries, Achilles tendinopathy (sometimes still referred to as Achilles tendinitis), it is important to report on the frequency of this particular injury in more detail (Table 2.2.).

Table 2.2 Frequency of Achilles tendinopathy in runners

Study	Total frequency (%)
Taunton et al., 2003	10
Taunton et al., 2002	5
Clement et al., 1981; Clement et al., 1984	6
Krissoff and Ferris,1979	18

2.2.3 Grading of the severity of running injuries

Overuse injuries in runners can also be classified according to injury severity. Injury severity can be progressively debilitating and can be measured through the following grades (Noakes, 2001a):

- Grade 1: The injury that only causes pain after exercise.
- Grade 2: The injury causes discomfort during exercises but in a severity not enough to cause reduction of training or racing performance.
- Grade 3: The injury causes pain during running which limits athletes training and performance.
- Grade 4: The injury is severe enough to prevent running.

Other authors have measured the severity of an overuse injury through time without training, and/or absence from work. In a survey of runners participating in a 16 km running race, it was found that 44% of the running injuries resulted in a cessation of training, 31% resulted in a medical consultation and 5% led to an absence from work (Marti et al., 1988).

2.2.4 General risk factors associated with running injuries

It is widely recognized that the aetiology of running injuries is multifactorial (van Mechelen, 1992; van Mechelen, 1995; Yeung and Yeung, 2001a; Hreljac, 2005; van Gent et al., 2007). Conventionally, the risk factors for running injuries can be divided into extrinsic and intrinsic factors. Extrinsic factors are detailed in Section 2.2.4.1 and intrinsic factors are explained in Section 2.2.4.2. Although, it has been suggested that this classification is artificial (Taimela et al., 1990), this remains a common method of classifying risk factors for injury, and will be used in this thesis (Taimela et al., 1990; Rolf, 1995; September et al., 2007). The general extrinsic and intrinsic risk factors for running injuries will now be reviewed.

2.2.4.1 Extrinsic risk factors associated with running injuries

The following extrinsic risk factors associated with running injuries will be reviewed in this section: training errors, running surface, lack of stretching, limited warm-up and cool-down, cross training, footwear - including running shoes.

2.2.4.1.1 Training errors

Training errors commonly refer to changes in running distance, frequency or intensity (van Mechelen, 1992; Hreljac, 2005), but in some cases, also refer to warm-up and cooling down, stretching exercises, training terrain and the use of incorrect equipment (van Mechelen, 1992; Sallade and Koch, 1992; McCrory et al., 1999). Training errors have consistently been related to running injuries (McKenzie et al., 1985; Lysholm and Wiklander, 1987; Sallade and Koch, 1992; Hreljac, 2005). From early case series, it has been suggested that more than 60% of running injuries could be attributed to training errors (James et al., 1978; McKenzie et al., 1985; Lysholm and Wiklander, 1987). The training variables that have been most often associated with injury are: high weekly training distance, increased running frequency, increased intensity of running, or a combination of these three training variables (van Mechelen, 1992; Yeung and Yeung, 2001a; Hreljac, 2005) . The relationship between each of these training variables and the risk of running injuries are now briefly reviewed.

Increased training distance

In one of the first studies where risk factors for running injuries were reported in a case series, it was estimated that excessive mileage could account for 29% of running injuries (James et al., 1978). In addition, training volume in the preceding month of an injury has been positively correlated (r=0.59) with injury risk (Lysholm and Wiklander, 1987). More recently, it has been shown that running distances of more than 64 km/week is a strong risk factor for the development of running injuries (van Gent et al., 2007).

In clinical studies, the injured population runs an average of 49 miles/week (James et al., 1978), but in other studies the average week distance was only 27 miles/week (Clement et al., 1981). The aforementioned reported variation on week training distance, led McKenzie et al. (1985) to question whether other related factors, rather than training distance alone, might explain injury incidence. More recently in an extensive meta–analysis, it has been shown that the incidence of running injuries is significantly lower

when running only for 15-30mins a day when compared with 45 mins a day (Yeung and Yeung, 2001a). Thus, increased running distance is seen to be an important risk factor for running injury: however, the exact weekly mileage that induces injury may vary according to the individual.

Increased training frequency

The relationship between increased running frequency and the risk of running injuries is less clear (van Mechelen, 1992; van Gent et al., 2007). Results of three prospective studies (Lysholm and Wiklander, 1987; Walter et al., 1989; Macera et al., 1989) indicate that increased running frequency is associated with an increased risk of injury. In contrast, no association between increased running frequency and injury risk was reported in one retrospective study (Marti et al., 1988). In a more recent systematic review (Yeung and Yeung 2001a), it was concluded that runners who train 1 to 3 days a week are less likely to be injured than those who train five days a week. Thus, increased running frequency may be associated with injury and training for more than 3 days a week might increase the injury risk.

Increased training intensity

The relationship between increased running pace (running intensity) and injury risk has only been explored in a small number of studies. In one retrospective study, running at a faster pace and racing more often were associated with injury in a questionnaire report of a 10km race (Jacobs and Berson, 1986). However, training speed was not associated with injury in another prospective study (Walter et al., 1989). Nevertheless, review studies have concluded that increased training intensity is an important variable that is associated with running injuries (McKenzie et al., 1985; Hreljac, 2005). Hrlejac (2005) associated the increased risk of running injury with the fact that higher speeds produce higher vertical impact forces. These increased forces will increase the magnitude of the load on the bones, joints and muscle structure, which will place the risk of injury at a higher position on the stress-frequency graph (Figure 2.5).

In summary, training errors, in particular increase in weekly training distance, is strongly associated with an increased risk of running injuries. The mechanism by which training errors may lead to an increased risk of running injury is related to increased frequency of loading and possible increased magnitude of loading (running speed) on the stress-frequency curve that was previously described in Figure 2.5.

2.2.4.1.2 Running surface

It is commonly believed by runners that different running surfaces may predispose to running injuries. There are many variables that could be considered as alterations in running surface. These include the hardness of the surface (concrete, gravel, grass, tar, sand, treadmill) and the gradient (uphill, downhill) of the surface (Nigg et al., 1995; Wank et al., 1998; Paradisis and Cooke, 2001; Pinnington et al., 2005). Despite this common assumption, there are limited number of scientific studies that have examined the possible association between surface and running injuries. In one prospective study, running on a concrete surface compared with running on asphalt has been associated with an increased risk of running injuries in female but not male runners (Macera et al., 1989). The reason why the incidence was higher amongst female runners on this surface could not be explained. In a case control study, there was a tendency that running injuries were more common if runners ran on crowned roads, and were performing hill training (Messier et al., 1991). However, in a retrospective study of entrants to a 10 km race, there was no relationship between running surface and injury (Jacobs and Berson, 1986). Evidence that running surface may be related to injury risk in runners has been reviewed, and it was concluded that running on a hard surfaces does not increase the risk of injuries when compared with running on soft surfaces (van Mechelen, 1992). Similarly, there is little evidence that hill running is associated with an increased risk of running injury (Cox, 1985).

2.2.4.1.3 Lack of Stretching

Although, a case control study found that injured runners were likely to incorporate stretching habits in their training routine (McCrory et al., 1999), most of the studies on injured prevention have found no support for the use of stretching before and after running in injury prevention (van Mechelen et al., 1993; Yeung and Yeung, 2001a). Hreljac et al. (2000) conclude that the use of stretching as part of warm-up and cool down may not be effective, but it is important to maintain the flexibility of the hamstring to prevent injury. This observation will be further explored in the section 2.2.4.2.8 Inflexibility.

2.2.4.1.4 Limited warm-up and cool-down

Active warm-up is an activity that results in a mild elevation of heart rate and ventilation but does not induce fatigue. Cool-down is the reverse of warm-up and is important to reduce heart rate and ventilation after exercise (McMurray, 1999). It has been suggested that limited warm-up and cool-down are risk factors for running injury. However, it has not been clearly established what is considered a limited warm-up or a limited cool down. In a single prospective study, it was documented that runners who never warm-up have a significantly higher risk of running injuries, compared with runners who sometimes, usually or always warm-up (Walter et al., 1989). In the same study, there was also no correlation between cool-down and running injury. Thus warm-up may be related to injury, however, there is a need for further studies in this area.

2.2.4.1.5 Cross training

Cross-training is defined as using another sport or technique to develop the performance in the main sport (Moran and McGlynn, 1997). It has been suggested that runners who engage in other sports activities (cross training) may have a decreased risk of injury (Moran and McGlynn, 1997). The main potential reasons for this is that by training for other sports, muscle strength imbalances may be improved, and that impact forces may

be reduced. This is particularly important if the other sports activities are non-weight bearing activities such as swimming, cycling, or rowing. However, in only very few studies has the relationship between injury risk and participation in other sports been reported (Clement et al., 1981; Walter et al., 1989; Taunton et al., 2003). In these studies, no association between cross training and running injury risk was reported.

2.2.4.1.6 Footwear

One of the most commonly frequently cited extrinsic risk factors for running injuries is the use of incorrect footwear or old ("worn") footwear. The relationship between footwear and running injury risk warrants in-depth analysis as it is an important component that was investigated in this thesis. This area is therefore, reviewed in detail in Section 2.4 (*A review of the biomechanics of running shoes*) of this thesis. The effect of insoles and orthotics will also be briefly reviewed in the same chapter section.

2.2.4.1.7 Summary: Extrinsic risk factors associated with running injuries – an evidence-based approach.

To provide a more clear and coherent view of the studies associated with running injuries in this literature review, the level of evidence of these studies was analysed using the evidence-based rating system adopted by the Journal of Bone and Joint Surgery (Obremskey et al., 2005). In this system, the levels evidence are depicted as ranging from Level I (good evidence) to Level V (poor evidence) (Appendix 1). Using these criteria, the levels of evidence for the extrinsic and intrinsic risk factors for injury are summarized in Table 2.3 and 2.4 respectively. The same criteria will also be used in subsequent sections in this thesis (2.3 *A Review of the Epidemiology of Achilles Tendinopathy* and 2.4. *A Review of Biomechanics of Running Shoes*).

Using evidence based approach; the main extrinsic risk factor associated with running injuries is training errors, more specifically training distance and possibly training intensity. There is insufficient or contradictory evidence that training frequency, running

surface, stretching, warm-up, cool-down and cross training are risk factors for running injuries.

Table 2.3. Extrinsic risk factors associated to running injuries using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Training errors	**A. Increase training distance**	
	Positive association:	
	Prospective studies: (Lysholm and Wiklander, 1987; Walter et al., 1989; Macera et al., 1989)	I
	Systematic review of Level 1 studies: (Yeung and Yeung, 2001a; van Gent et al., 2007)	I
	Retrospective studies: (James et al., 1978)	II
	Systematic review of Level II studies: (Hreljac, 2005)	II
	Expert opinion: (Sallade and Koch, 1992)	V
	No association:	
	Expert opinion: (McKenzie et al., 1985)	V
	B. Increase training frequency	
	Positive association:	
	Prospective studies: (Lysholm and Wiklander, 1987; Walter et al., 1989; Macera et al., 1989)	I
	Systematic review of Level I studies: (Yeung and Yeung, 2001a)	I
	Systematic review of Level II studies: (van Mechelen, 1992)	II
	No association:	
	Retrospective study (Marti et al., 1988)	II
	C. Increase training intensity	
	Positive association:	
	Retrospective study: (Jacobs and Berson, 1986)	II
	Systematic review of Level II studies: (Hreljac, 2005)	II
	Expert opinion: (McKenzie et al., 1985)	V
	No association:	
	Prospective study: (Walter et al., 1989)	I
	Systematic review of Level 1 studies: (van Gent et al., 2007)	I

Table 2.3 continued. Extrinsic risk factors associated to running injuries using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Running on hard surface	**Positive association:**	
	Prospective studies female runners (Macera et al., 1989)	I
	Case control (Messier et al., 1991)	III
	No association:	
	Prospective study male runners (Macera et al., 1989)	I
	Retrospective studies (Jacobs and Berson, 1986)	II
	Systematic review level II studies (van Mechelen, 1992)	II
Lack of Stretching	**Positive association:**	
	Systematic review of Level II studies: (Hreljac, 2005)	II
	Case control (McCrory et al., 1999)	III
	No association:	
	Systematic review level I studies (Yeung and Yeung, 2001a)	I
	Systematic review level II studies (van Mechelen, 1992)	II
	Case control (van Mechelen et al., 1993)	III
Limited Warming – up and cooling -down	**A. Warming-up**	
	Positive association:	
	Prospective study (Walter et al., 1989)	I
	B. Cooling - down	
	No association:	
	Prospective study (Walter et al., 1989)	I
Cross-training	**No association:**	
	Prospective studies (Walter et al., 1989; Taunton et al., 2003)	I
	Retrospective study (Clement et al., 1981)	II

2.2.4.2 Intrinsic risk factors for running injuries

A number of intrinsic factors have been associated with running injuries and the following intrinsic factors will be reviewed in this section: history of previous injury, less running experience, age (older or younger), gender, increase body height, mass and body mass index (BMI), biomechanical factors, variability in biomechanical parameters, abnormal limb abnormalities, inflexibility, and muscle weakness.

2.2.4.2.1 A history of a previous running injury

A history of a previous running injury has consistently been reported in several epidemiological studies as a risk factor for a running injury (Walter et al., 1989; Macera et al., 1989; Macera et al., 1991; Taunton et al., 2003; van Gent et al., 2007). In a prospective cohort study, it was observed that runners who were injured in the previous year had a 50% higher risk of becoming injured again in the following year (Walter et al., 1989). Similarly, in another study it was found that the risk of having a running injury was significantly increased if there was a history of a previous injury (odds ratio of 2.7) (Macera et al., 1989). Furthermore, in another retrospective study, marathon runners who reported an injury a year before a marathon race, had a significantly increased chance of developing a musculoskeletal injury after a marathon (Macera et al., 1991). In only one retrospective study, no relationship between a history of previous running injuries and the risk of developing a new running injury was reported (Taunton et al., 2002).

The results of most of these studies strongly support the relationship between a history of previous injury and the increased risk of developing a running injury in future. The precise reasons why a past injury is a strong predictor of a new injury are not known. It has been suggested that this could be related to an incomplete healing of the original injury, individual inherent predisposition to injury, or even an uncorrected biomechanical problem (Macera, 1992).

2.2.4.2.2 Less running experience

There is some evidence suggesting that less running experience is associated with an increased risk of developing a running injury (Macera et al., 1989; van Gent et al., 2007). Less running experience is usually defined in relation to years of participating in running. However, interpretation of findings from studies is difficult because this definition is not uniformly applied as the experience is not always only in running but sometimes refers to other sporting activities, and the relationship between running experience and injury may not apply to all types of running injuries (Macera et al., 1989). Furthermore, the methodological quality of studies varies and includes retrospective case series, prospective cohort studies and systematic reviews (Walter et al., 1989; Macera et al., 1989; Satterthwaite et al., 1999; Taunton et al., 2002; van Gent et al., 2007; Fredericson and Misra, 2007).

It has been suggested from retrospective studies that runners, who were active for less than 8.5 years, were 2.5 times more likely to develop a running injury (Taunton et al., 2002). However, the sports activities reported were general and were not always related to running. In line with this suggestion, a retrospective study found that running for less than 3 years increases the risk of developing a running injury (Macera et al., 1989). Also in support of these findings is a review article concluded that more experienced runners were less prone to injury, and that the number of years running is inversely related to the incidence of injury (Fredericson and Misra, 2007). However, a systematic review revealed a diverse response, according to the injury type (van Gent et al., 2007). Hamstring and knee injuries were associated with inexperienced runners, while foot injuries were associated with more experienced runners. Conflicting results were also found in prospective studies where running experience was not associated with injury risk in one study (Walter et al., 1989), but in a another prospective study (Satterthwaite et al., 1999) runners who have never run a marathon, had more than 50% increased risk of injury before they run their first marathon.

In conclusion, although retrospective studies suggest that less running experience is associated with injury, prospective studies and systematic reviews have found conflicting evidence. Therefore, it is inconclusive whether running experience is associated with injury.

2.2.4.2.3 Age

It has been suggested that older age as well as a very young age are risk factors for running related injuries. Older age may be related to an increased risk because of reduced flexibility and muscle strength, and reduced capacity of tissue regeneration. In another hand, younger age may predispose to an increased risk because of relative inexperience, and possibility to run at a higher intensity (faster pace).

The findings of studies on the effect of age on incidence of running injuries are contradictory. For example, a study based on a race survey, found a significant decrease of running injuries with increasing age (Marti et al., 1988). Supporting this finding, a retrospective study found that being less than 34 years old is a risk factor in developing patellofemoral pain in both genders (Taunton et al., 2002). The same study found that illiotibial band syndrome, patellar tendinopathy and tibial stress syndrome are significantly more common in male runners younger than 34 years old. However, the same group of authors have found in a more recent prospective study, that women over 50 years of age have a higher risk of developing a running injury (Taunton et al., 2003). In accordance with this finding, runners with hamstring injuries were older than those without the injury (Wen et al., 1997). There are other studies, which have not found a relationship between age and the incidence of running injuries (Jacobs and Berson, 1986; Walter et al., 1989; Macera et al., 1989).

Presently even prospective studies are contradictory in their findings showing positive, negative or no association between older age and injury risk. Thus, it can be concluded after analysing the literature that there is not a clear relationship between age and injury.

2.2.4.2.4 Gender

The risk of developing a running injury may also be related to gender. Although this relationship has not been studied extensively, it also appears that certain types of running injuries may be more common in one gender compared with the other.

For instance, according to two retrospective studies, Achilles tendinopathy tends to be more common in males than females runners (7.9% males and 3.2% females) (Clement et al., 1981), while patellofemoral pain appears to be more common among female runners (10.3% females and 6.1% males) (Taunton et al., 2002). However, when different types of injuries have been studied together, it has been found that running injury and gender are not related (Jacobs and Berson, 1986; Lysholm and Wiklander, 1987; Walter et al., 1989). Hence, there is strong evidence that gender might not be related to injury risk in general. However, this observation might be different if the analysis is ascribed to a certain type of injury.

2.2.4.2.5 Increase height, mass and body mass index (BMI)

Anthropometric variables such as height and body mass might be associated with injuries due to a greater load on bones, joints and connective tissues (van Mechelen, 1992). However, the higher load might be balanced by stronger muscles and larger bone surface. Therefore, the anthropometrics factors in this section are divided into height, mass and body mass index factors, and will be discussed separately.

Height

In one multivariate analysis (Taunton et al., 2002), male runners smaller than 1.57 m were at a higher risk in developing plantar fasciitis. Contradicting this finding, a cohort study has shown that increased height is related to injury risk (Walter et al., 1989). According to this study, runners taller than 1.70m had a significantly greater risk of injuries. Thus, there is insufficient evidence to draw definitive conclusions on the associating between height and running injury risk.

Body mass

In one study, it was reported that women with a body mass less than 60kg are less prone to develop plantar fasciitis (Taunton et al., 2002) while in another cohort study body mass was not related to running injury (Walter et al., 1989).Therefore, similar to height, there is not enough evidence to associate body mass with running injury risk.

Body mass index

Body mass index (BMI) is the relationship between body mass and height. In one retrospective study female runners with a BMI less than 21kg/m^2 had a higher risk of developing a tibial stress fracture and spinal injuries (Taunton et al., 2002). Similar results were found by the same group of authors in a prospective study, where participants were involved in similar training programme. In this study, male runners with a BMI greater than 26 kg/m^2 had a reduced risk of injuries (Taunton et al., 2003). In order to explain this finding, it has been suggested that runners with a lower body mass may have insufficient lean body mass to compensate for the biomechanical stresses involved during running (Neely, 1998; Taunton et al., 2002). This argument could also explain why women with lower BMI who have lower levels of estrogen, and therefore lower bone density, have an increased the risk of injury (Taunton et al., 2002). However, in two prospective studies there was no association between BMI and injury risk in runners (Walter et al., 1989; Macera et al., 1989).

Thus, although the literature presents conflicting results concerning the association of body mass, height and injury risk, increased BMI appears to be protective against injury according to some recent prospective and retrospective studies. Although some older prospective studies dispute this as no association was found between BMI and injury.

2.2.4.2.6 Biomechanical factors

It has been suggested that biomechanical factors such as increased foot pronation and impact forces are associated with running injuries.

Increased foot pronation

As explained previously (section 2.1.4.5 *Subtalar joint pronation and supination*), subtalar joint pronation occurs during early and midstance phase and reduces the level of early impact forces (Novacheck, 1998b; Hreljac, 2005). It has been suggested that if subtalar joint pronation is excessive or prolonged after midstance it could produce larger torques and generate instability, which in turn could be related to injury (Hreljac, 2005). Additionally, it is possible that it is not the amount of eversion that can be related to injury but the way that eversion is transferred to tibial rotation, considering that similar values of eversion can generate different values of tibia rotation between individuals (Hintermann and Nigg, 1998). It has also been speculated that if maximum pronation and maximum knee flexion do not occur simultaneously, conflicting torsional forces will be generated through the tibia, which may lead to injuries (Hintermann and Nigg, 1998). This tends to occur with the increase in speed and obstacle height, which increases the ground reaction forces and the asynchrony between the actions of the ankle (pronation and supination) and the knee (flexion and extension) (Stergiou et al., 1999).

The study of McClay and Manal (1998) reported the three dimensional biomechanical differences between excessive pronators and normal pronators runners who were screened by 2D analysis. In this study it was found that runners, who pronate, had a twofold increase in peak eversion and increase of eversion at heel strike; additionally the foot was in an everted position at toe-off instead of the expected inverted position of a neutral stride (McClay and Manal, 1998). Also, there was significantly higher knee flexion at initial contact, and rearfoot dorsiflexion and eversion velocity during stance phase in this group. The authors suggested that the increase in angular velocity that was observed in the pronator group could cause the muscles to become overworked, which could result in an overuse injury (McClay and Manal, 1998).

Clinical studies more often associate pronation with overuse injury (James et al., 1978; McKenzie et al., 1985; Subotnick, 1985). In these studies pronation was established through static measurements. Although, there is evidence that static measurements can be applied to estimate pronation during walking gait (Torburn et

al., 1998), there is still limited support from the literature in relation running biomechanics and further studies need be conducted.

According to clinical studies, runners with mild to severe tibial and rearfoot varus alignment had excessively pronation of the ankle and subtalar joints during the stance phase on running (Clement et al., 1981). It is expected that due to an increase of the transverse plane motion of the lower limb, runners with excessive pronation may have developed running injuries. One of the first studies to relate pronation to injury dates from 1978 (James et al., 1978). The authors noted that from a population of 180 injured runners who were assessed using static measurements, 58% of runners had low arch foot, which they associated with pronation, 20% had high arch feet and 22% present normal arch feet. The runners with a low arch foot presented with the following injuries: medial tibial stress syndrome, plantar fasciitis, Achilles tendinitis and knee pain. Several later clinical or review studies and expert opinion papers have also related pronation to running injury (Clement et al., 1981; McKenzie et al., 1985; Rolf, 1995).

In one of the earliest case control studies in which biomechanical parameters were studied in a group of uninjured runners and runners with injuries (shin splint, plantar fasciitis and IT band friction syndrome), it was found that maximum pronation and maximum pronation velocity were significant discriminators for shin splints but not for the other injuries (Messier and Pittala, 1988). However, opposing this finding, a more recent case control study found out that uninjured runners had greater pronation velocity compared with runners who sustained an injury (Hreljac et al., 2000). Along the same lines, another case control study has found that runners with illiotibial band syndrome have a tendency to pronate significantly less than uninjured runners and present a significantly higher maximum supination velocity (Messier et al., 1995). Finally, in another case control study there were no differences in rearfoot kinematics between runners who had patellofemoral pain syndrome and uninjured runners (Messier et al., 1991).

In a more recent prospective study (Willems et al., 2007), the running biomechanics of 400 physical education students was examined to determine injury risk factors associated with exercise-related lower leg pain (shin splints, shin pain, medial tibial

stress syndrome, periostitis, compartment syndrome and stress fractures). In this study it was found that the group, which developed an injury, had an increased pronation excursion, a delay in maximal eversion and an accelerated reinversion compared with the group that did not develop an injury.

Although there is a recent prospective study, which supports the association of pronation with injury, other studies with different level of clinical evidence present contradicting findings (Table 2.4). Therefore, it can be concluded that pronation might be associated with injury, but there is a need for further prospective studies, and a clearer definition and measurement of subtalar joint pronation parameters.

Increased ground reaction forces

It has been suggested that increases in impact forces will increase the magnitude of stress that is applied on the tissue and therefore increase the risk of injury (Figure 2.5) The two common kinetic variables that have been associated with a increased risk of running injuries are increased vertical impact force and vertical loading rate (Cavanagh and Lafortune, 1980).

In one of the early case control study it was documented that runners with patellofemoral pain had a greater vertical propulsive force even when running at a slower running speed than uninjured runners in a control group (Messier et al., 1991). Likewise, it has recently been documented that instantaneous and average vertical loading rate were significantly higher among female runners who had a history of tibial stress fracture (Milner et al., 2006). In this same study, the horizontal braking force peak was significantly lower, and occurred later on the tibial stress fracture group. Supporting the findings of vertical forces and injury, another study has found that uninjured runners presented lower impact forces and loading rates compared to injured runners (Hreljac et al., 2000). However a study with runners with iliotibial band syndrome (ITBS) did not find significant differences on vertical impact forces, though horizontal braking force peak was significantly lower in the ITBS group (Messier et al., 1995).

In summary, most of the studies support the theory that runners who are injured presented higher impact forces during running. However, there is little evidence to suggest that this is a cause-effect relationship as there is a lack of prospective studies in this area.

2.2.4.2.7 Variability in biomechanical parameters

Variability in biological parameters is inherent within and between any biological systems (Newell and Corcos, 1993). According to Hamill et al. (1999) the notion of variability pattern might be functional and not considered simply to be "noise". Variability has been studied in several sports, and in medical disciplines. For example, in cardiology, a decrease in heart rate (beat-to-beat) variability has been associated with cardiac pathology (Huikuri et al., 1999). And in sports science, overtrained athletes have shown a decrease in heart rate variability during standing (Uusitalo et al., 2000). In the context of biomechanical variability, the variability of certain parameters (kinetic, kinematic and muscle activity) can also be studied. Variability in these parameters can be studied 1) within participants (stride to stride) and this is referred to as intra-participant variability, or 2) between participants and will be referred to as or inter-participant variability (Bartlett et al., 2007).

Intra-participant variability of biomechanical parameters

It has been suggested that intra-participant (or stride to stride) variability may be important to alternate the load on the joints from stride to stride (Hamill et al., 1999; Heiderscheit et al., 1999).

As already indicated, running kinematics and muscle activity may be altered from stride to stride during running. This mechanisms may be important to regulate the magnitude of the impact force during running and therefore muscle vibration, which can increase muscle and tendon load (Nigg and Wakeling, 2001; Hardin et al., 2004). It has been postulated that sensory feedback on the sole of the foot is an important mechanism to determine movement variability (Kurz and Stergiou, 2003). Therefore, input signal from impact forces could alter subsequent movement (Chen et al., 1995; Nurse and Nigg, 2001). In support of this concept, is the observation that a larger

stride to stride variability was documented in runners when they ran in a barefoot condition rather than when they ran in a shod condition (Kurz and Stergiou, 2003). It was suggested that the reduction of variability when running with shoes indicated a movement action in a narrower range from stride to stride, and this may restrict the adaptation to different external inputs.

The reduction in intra-participant (stride-to-stride) variability may be associated with injury as runners with patellofemoral pain (PFP) showed less variability between the joint segment motions than uninjured runners in a case-control study (Hamill et al., 1999). Similarly, in another case control study, lower joint coordination variability was found in runners with PFP at heel strike compared with uninjured runners (Heiderscheit et al., 2002). However, the authors did not find significant differences in the average data across the entire running cycle. Supporting this latest finding, in another case control study no significant differences were found on joint coupling pattern when runners with different types of injuries were treated successfully with orthotics and were compared to uninjured runners (Ferber et al., 2005).

Therefore, it appears that decreased intra-participant (stride-to-stride) variability may be associated with an increased risk of running injury, however this requires further investigation. It is also of interest to note that intra-participant variability seems to increase with the increase on external sensory input signal (e.g. running with shoes vs. running in barefoot) in an uninjured population.

Inter-participant variability

Inter-participant variability refers to the variability biomechanical parameters between groups (e.g. injured vs. uninjured runners). Inter-participant variability has been investigated in runners using different shoes and this will be further discussed in Section 2.4.3.2 *Effect of shoe proprieties on running biomechanics.* However, to our knowledge there are no studies which have investigated inter-participant variability in an injured population.

Therefore, it can be concluded that although there are some evidence that intra-participant (or stride-to-stride) variability is reduced in an injured population (Hamill

et al., 1999; Heiderscheit et al., 2002), this may still require further investigation as there are some contradictory observations that vary according to the phase of running cycle investigated and the type of injury (Heiderscheit et al., 2002; Ferber et al., 2005). Furthermore, inter-participant variability of biomechanical parameters as possible risk factors for running injuries has, to our knowledge, not been investigated.

2.2.4.2.8 Abnormal lower limb alignment

Lower limb, anatomical or anthropometric variables have often been related to running injuries (McKenzie et al., 1985; Lysholm and Wiklander, 1987; Brunet et al., 1990; McCaw, 1992; Kvist, 1994). Their association with injuries can be related to the fact that anthropometric abnormalities may cause unfavourable biomechanics, which can induce injury. More specifically, anthropometric abnormalities may affect external forces (e.g. ground reaction forces) or internal forces (e.g. stress imposed on tissues).

There are several anthropometrical variables, which have been related to increased injury risk in runners. These include: leg-length discrepancy, height of longitudinal arch and lower extremity alignment. One of the early studies, which attempted to analyse the cause of running injuries retrospectively, found that 40% of the injuries could be related to at least one lower limb misalignment (Lysholm and Wiklander, 1987).

Skeletal imbalances such as leg length could alter the pattern of mechanical stress within a joint. Some retrospective studies have reported an increased in injury rate in runners with leg length inequalities over runners without this characteristic (Brunet et al., 1990; McCaw, 1992). However, other case control studies and retrospective studies did not find leg length discrepancies to be a significant factor between injured runners and uninjured runners (Gross, 1983; Messier et al., 1991). Therefore, it can be concluded that leg length inequality might not be associated with injuries but there is a need for further prospective studies.

Foot type has also been suggested to play a role in the development of a running injury (McKenzie et al., 1985). It is suggested that runners with pes planus are more

capable of force dissipation due to the increased ground contact. However they tend to suffer from injuries related to increased motion of the subtalar joint (increased pronation) (McKenzie et al., 1985). Runners with cavus foot on the other hand, show a decrease of subtalar motion and therefore, have decreased in shock absorption. As mentioned earlier (Section 2.2.4.2.6 *Biomechanical factors*), the transfer between foot eversion and tibia rotation has been associated with running injuries. In this regard Nigg et al. (1993) found that individuals with high arches had a greater movement transfer from pronation to tibia rotation than individuals with a low foot arch (Nigg et al., 1993). Therefore, according to the results of this study, individuals with high foot arches are probably at a higher risk of developing an overuse injury.

The results of epidemiological studies where the relationship between foot type and injury risk have investigated are also not clear. Foot type, as measured by the arch index (Cavanagh and Rodgers, 1987), was similar between uninjured runners and runners with patellofemoral pain (Messier et al., 1991). In studies where different injuries were analysed together, high arch feet was not considered a risk factor when runners were observed prospectively to the incidence of injury (Montgomery et al., 1989; Wen et al., 1997; Lun et al., 2004). Therefore, as high clinical evidence-based studies have not found an association between foot type and injury, it can be concluded that this is not an aetiological factor.

Other than foot type, other anatomical measurements have been associated with overpronation and therefore injury. Some examples of these anatomical measurements are: navicular drop, Q angle, tibia vara and forefoot varus (Messier et al., 1991; Hintermann and Nigg, 1998; Cornwall and McPoil, 1999). Navicular drop has been considered a valid measure of subtalar motion during walking gait (Cornwall and McPoil, 1999), but it was not related to subtalar motion during landing (Hargrave et al., 2003). However, as far as we know, the navicular drop and subtalar motion during running not been studied. In epidemiological prospective studies, no association was found between navicular drop and the general incidence of injury with collegiate cross country runners was found (Reinking et al., 2007). However, according to one prospective study, navicular drop test measurements can correctly predict the incidence of 76% of runners, who will develop medial tibial stress syndrome (Bennett et al., 2001). However, a more recent prospective study has not found an association

between navicular drop and medial tibial stress syndrome (Bandholm et al., 2008). It can be therefore concluded that there is some evidence that there is an association between navicular drop and incidence of injury in general. However, further studies with specific running injuries should be developed to clarify some contradictions in the literature.

The quadriceps (Q) angle is another anthropometric variable that has constantly been associated with increased pronation ad increased risk of running injury. An increased Q angle was related to patellofemoral pain in one case control study (Messier et al., 1991). It was found that Q angle in the range of 15 to 20° was associated with injury. Additionally, in a prospective study, runners with Q angle above 20° were more prone to develop knee injury, while runners with Q angle less than 4° had a higher propensity to develop shin injury (Rauh et al., 2007). Therefore, data from both retrospective and prospective studies have shown that Q angle may be associated with injuries, in particular the knee. However, the relationship between Q angle and other running injuries requires further investigation.

A prospective cohort study has found that anthropometrical variables such as femoral neck anteversion, knee and patella alignment and rearfoot valgus are significantly related to specific injuries (Wen et al., 1997). A later prospective study examined 87 recreational runners for static lower limb alignment (Lun et al., 2004). These runners were observed for any running related injury for a period of six months. The study concluded that static alignment was not related to lower limb injury in general, but static lower limb alignment could be injury specific, as runners with patellofemoral pain syndrome presented significantly different values of right ankle dorsiflexion and right knee varum and left foot forefoot varus. This result needs to be treated with caution as the population with injured knees consisted of only 6 runners (Lun et al., 2004).

In summary, there are clearly some disagreements in the literature between the relationship of anatomical variables and overuse injury. The controversial findings of the studies are perhaps related to differences in measuring techniques, and also to the fact that anatomical parameters may not cause alteration in running biomechanics variables.

2.2.4.2.9 Inflexibility

Flexibility is defined by the ability to move a joint through its complete range of motion (American College of Sports Medicine, 2000). It has been postulated that running overuse injury may result from muscle shortening, due to muscle tiredness or limited muscle strength, which could increase stress on the muscle joints (van Mechelen, 1992). This can also be indicative of muscular imbalance, which can induce inappropriate biomechanics, although, there is very limited literature on this aspect (Hreljac et al., 2000).

In a case control study, injured runners who sustained at least one overuse running injury presented lower flexibility as measured by the sit and reach test when compared with uninjured runners (Hreljac et al., 2000). According to the researchers, limited flexibility may be associated with an increase of muscle stiffness. On the contrary, in a retrospective study injured runners were found to present a greater range of motion (ROM) of plantarflexion when compared with uninjured runners (Warren and Jones, 1987). However, in another case control study, which investigated the anthropometrical measurements of runners with patellofemoral pain (Messier et al., 1991), ROM of plantarflexion and ROM of knee flexion were not associated with injury. Additionally, another case control study has found that ROM of hip was limited but ROM of the ankle was similar in runners with different types of injury (van Mechelen et al., 1992).

In summary, it is difficult to draw any conclusions about the effect on inflexibility on risk of injury. The results appear to vary according to the muscle tested and the injury type. Further studies with specific injuries and muscles and also prospective studies need to be conducted.

2.2.4.2.10 Muscle weakness

An imbalance in strength between agonist and antagonist muscles has been suggested as a risk factor in the development of a running injury (Clement et al., 1984; Subotnick, 1985). The association of muscle strength and injury is still under

investigation as there are a limited number of studies in literature and no prospective investigations.

In one case control study, no relationship was found between knee flexion-extension peak forces and patellofemoral pain (Messier et al., 1991). Although the same study found a significant difference in muscle endurance, as runners with patellofemoral pain had weaker knee extensors, but stronger knee flexors. Runners with iliotibial band syndrome (ITBS) had a weaker hip abduction strength compared with uninjured runners, and also when compared with their unaffected limb (Fredericson et al., 2000). Likewise, in another more recent study, which has also investigated hip muscles' strength on runners with multiple injuries, found that injured runners had stronger hip adductors and weaker hip abductors and flexors on the injured side compared with the uninjured side (Niemuth et al., 2005). Whilst this may not be a cause-effect relationship, the strengthening exercises to improve balance and weakness of the hip muscles may be an advantage in the treatment of running injuries in general.

In summary, there is limited evidence that muscle imbalances and weakness may be associated with increased risk of running injuries. However, there is some evidence suggesting that weaker hip abductors may be associated with a specific running injury, the ITBS.

2.2.4.2.11 Summary: Intrinsic risk factors for running injuries – an evidence-based approach

The outline of the reviewed studies on the intrinsic risk factors associated with running injury show that a history of previous injury is strongly associated with injury (Table 2.4). Although one study has found no association between previous injury and future injuries (Taunton et al., 2002), quality systematic review and prospective studies have clearly established a relationship between both (Walter et al., 1989; Macera et al., 1989; Taunton et al., 2003; van Gent et al., 2007). However, there is limited or contradictory evidence for the relationship between running injury risk and intrinsic risk factors such as less running experience, age, gender, anthropometrics

(height, mass and BMI), biomechanics (pronation and impact force), variability of biomechanical parameters, lower limb alignment, inflexibility and muscle weakness.

Table 2.4. Intrinsic risk factors associated to running injuries using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
History of previous injury	**Positive association:**	
	Prospective studies: (Walter et al., 1989; Macera et al., 1989; Taunton et al., 2003)	I
	Systematic review of Level I studies: (van Gent et al., 2007)	I
	Retrospective studies: (Macera et al., 1991)	II
	No association:	II
	Retrospective study: (Taunton et al., 2002)	
Less running experience	**Positive association:**	
	Prospective study: (Satterthwaite et al., 1999)	I
	Systematic review of Level I studies: (van Gent et al., 2007)	I
	Retrospective study: (Macera et al., 1989; Taunton et al., 2002)	II
	Systematic review of Level II studies: (Fredericson and Misra, 2007)	II
	No association:	I
	Prospective study (Walter et al., 1989)	I
	Systematic review of Level I studies: (van Gent et al., 2007)	
Age	**Negative association:**	
	Prospective study: (Satterthwaite et al., 1999)	I
	Retrospective studies: (Marti et al., 1988; Taunton et al., 2002)	II
	Positive association:	
	Prospective study: (Taunton et al., 2003)	I
	Retrospective studies: (Wen et al., 1997)	II
	No association:	
	Prospective study: (Walter et al., 1989; Macera et al., 1989)	I
	Retrospective study: (Jacobs and Berson, 1986)	II

Table 2.4 continued. Intrinsic risk factors associated to running injuries using evidence-based medicine (EBM) criteria(Obremskey et al., 2005).

Risk factor	Study information	Level of Evidence (I-V)
Gender	**No association:**	
	Prospective studies: (Lysholm and Wiklander, 1987; Walter et al., 1989)	I
	Retrospective study: (Jacobs and Berson, 1986)	II
	Positive association with specific gender and injury type:	
	Retrospective study: (Clement et al., 1981; Taunton et al., 2002)	II
Increase height, body mass, and BMI	**A. Increased Height:**	
	Positive association:	
	Prospective study: (Walter et al., 1989)	I
	No association:	
	Retrospective study: (Taunton et al., 2002)	II
	B. Increased Mass:	
	Positive association:	
	Retrospective study: (Taunton et al., 2002)	II
	No association:	
	Prospective study: (Walter et al., 1989)	I
	C. Increased BMI	
	Negative association:	
	Prospective study: (Taunton et al., 2003)	I
	Retrospective study (Taunton et al., 2002)	II
	No association:	
	Prospective study: (Walter et al., 1989; Macera et al., 1989)	I

Table 2.4 continued. Intrinsic risk factors associated to running injuries using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Biomechanical factors	**A. Increased foot pronation:**	
	Positive association:	
	Prospective study: (Willems et al., 2007)	I
	Retrospective studies: (James et al., 1978; Clement et al., 1981)	II
	Case control: (Messier and Pittala, 1988)	III
	Expert opinion: (McKenzie et al., 1985; Subotnick, 1985; Rolf, 1995)	V
	No association:	
	Case control: (Messier and Pittala, 1988; Messier et al., 1991)	III
	Negative association:	
	Case control: (Messier et al., 1995; Hreljac et al., 2000)	III
	B. Increased vertical impact forces:	
	Positive association:	
	Case control: (Messier et al., 1991; Hreljac et al., 2000; Milner et al., 2006)	III
	No association:	
	Case control: (Messier et al., 1995)	III
Variability in biomechanical parameters	**A. Intra participant variability**	
	Positive association:	
	Case control: (Hamill et al., 1999; Heiderscheit et al., 2002)	III
	No association:	
	Case control: (Heiderscheit et al., 2002; Ferber et al., 2005)	III
	B. Inter participant variability	
	No evidence	

Table 2.4 continued. Intrinsic risk factors associated to running injuries according to evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Abnormal lower limb alignment	**Positive association:**	
	Prospective study: (Lysholm and Wiklander, 1987; Wen et al., 1997; Bennett et al., 2001; Rauh et al., 2007)	I
	Retrospective study: (Brunet et al., 1990)	II
	Case Control: (Messier et al., 1991)	III
	Expert opinion: (McKenzie et al., 1985; McCaw, 1992; Kvist, 1994; Hintermann and Nigg, 1998)	V
	No association:	
	Prospective studies: (Montgomery et al., 1989; Wen et al., 1997; Lun et al., 2004; Reinking et al., 2007; Bandholm et al., 2008)	I II
	Retrospective study (Gross, 1983)	III
	Case Control (Messier et al., 1991)	
Inflexibility	**Positive association:**	
	Case control: (van Mechelen et al., 1992; Hreljac et al., 2000)	III
	Negative association:	
	Retrospective study: (Warren and Jones, 1987)	II
	No association:	
	Case control: (Messier et al., 1991; van Mechelen et al., 1992)	III
Muscle weakness	**Positive association:**	
	Case control: (Fredericson et al., 2000; Niemuth et al., 2005)	III
	No association:	
	Case control: (Messier et al., 1991)	III

2.2.4.3 Summary: Risk factors for running injuries

Taking all the above aetiological factors into consideration, running injuries may be prevented if the training load, or more specifically training volume, progresses gradually. However, each individual may have a specific threshold above which he/she may be more prone to injury. Furthermore, it is important for the runner to fully recover from an injury before returning to training.

2.3 A Review of the Epidemiology of Achilles Tendinopathy

2.3.1 Introduction

It has been shown in a number of studies that 5-18% of all running injuries are due to Achilles tendinopathy, which makes it one of the more common causes of running injuries (Krissoff and Ferris, 1979; Clement et al., 1984; Lysholm and Wiklander, 1987; Taunton et al., 2002). It has also been suggested that Achilles tendon injury may account for 17% of all sporting injuries (McLauchlan and Handoll, 2001). In a report of 3,336 patients from a sports medicine clinic (90% of whom were runners or were involved in a sport activity that involves running), 455 (14%) of these patients had Achilles tendon problems (Kvist, 1994).

A chronic Achilles tendon injury can be considered a serious injury as in a group of competitive track and field runners who had Achilles tendon injuries, 16% were forced to abandon their sport, and only 54% continued to compete but with discomfort (Welsh and Clodman, 1980).

Although the frequency of Achilles tendon injuries among runners is still high, there appears to be a reduction in its incidence over the past three decades. In 1979, it was reported that Achilles tendinopathy accounted for 18% of the total running injuries (Krissoff and Ferris, 1979). More recently in a retrospective case control study, Achilles tendon injury accounted for only 4.7% of the total running injuries (Taunton et al., 2002) (Table 2.1). It has been postulated that the increased awareness of importance of gastrocnemius flexibility and selection of a better designed running

shoes could have contributed to a possible reduction in this type of injury (Clement et al., 1984).

The focus of this thesis is on Achilles tendinopathy in runners. Therefore, a brief review of the anatomy of the Achilles tendon is warranted, and this will be followed by a review of the biomechanics of the Achilles tendon.

2.3.2 Anatomy of the Achilles tendon

A detailed discussion of the anatomy of the Achilles tendon is beyond the scope of this thesis. However, aspects of the anatomy of the Achilles, tendon, particularly in relation to the biomechanical properties of the tendon, will be briefly reviewed.

The Achilles tendon is the largest and strongest tendon in the human body. The gastrocnemius and soleus muscles, which are collectively known as triceps surae, insert into the Achilles tendon. The prime function of these muscles is plantar flexion of the ankle. The gastrocnemius has its origin from the lateral and medial femoral condyle, while the soleus originates from posterior surface of the tibia and fibula. The tendinous part of the triceps surae has a poor blood supply, particularly in the portion that is 2 to 6 cm above the insertion of the Achilles tendon into the calcaneus. This is also the area that is more susceptible to injury (Clement et al., 1984; Jones, 1998). However, vascularisation in other areas of the tendon are evenly distributed (Astrom and Westlin, 1994a). Furthermore, blood flow seems to increase in symptomatic Achilles tendons (Astrom and Westlin, 1994b).

The Achilles tendon fibres rotate laterally as they descend from proximal to distal. There are three patterns of rotation. In the most common pattern, the gastrocnemius contributes to two thirds of the fibres and the soleus contributes to one third. In the second most common pattern, the contribution between gastrocnemius and soleus are evenly distributed (about half each). In the third and least common pattern, the soleus is responsible for two thirds and the gastrocnemius for the remaining one third (Cummins et al., 1946). This rotation of the Achilles tendon fibres may play an

important role in the development of pathologic conditions of the Achilles tendon (Jones, 1998).

Histologically, the Achilles tendon consists of 30% of collagen, 2% elastin and 68% water (Novacheck, 1998a). With aging, the production of enzymes necessary for collagen formation are reduced, the tissue takes longer to repair, the tissue becomes less elastic and tensile strength is reduced. All these effects result in a stiffer tendon and one that is more likely to tear (O'Brien, 1992). It has been shown that younger tendons (<35 years old) have a higher maximum rupture force and a lower stiffness compared with older tendons (>35 years old) (Thermann et al., 1995). On the other hand, training can increase the tensile and maximum static strength of the tendon by increasing collagen synthesis, the number and size of fibrils and the concentration of metabolic enzymes (O'Brien, 1992). The Achilles tendon does not have a synovial sheath like other tendons in the body, instead it is enveloped by membrane consisting of two layers which is called the paratenon (Kader et al., 2002).

2.3.3 Biomechanics of the Achilles tendon

2.3.3.1. Introduction

The study of the biomechanics of the Achilles tendon is directly related to the capacity that this tissue has to accommodate a load of high magnitude as well as repeated loads. In this section the general mechanical properties of Achilles tendon will be reviewed in combination with the biomechanical response of this tissue during running.

2.3.3.2. General mechanical properties of the Achilles tendon

The tendon loses its wavy configuration when it is stretched more than 2%. As it deforms, the fibres respond linearly to increasing tendon loads (O'Brien, 1992). The tendon can stretch up to 4% without damage (Kader et al., 2002). The overuse mechanical theory of tendon disorders suggests that the tendon cannot sustain constant strain at a rate between 4-8%, after which rupture may occur. In the case when the tendon becomes fatigued because of the repetitive stress, it loses the ability

of the cells to repair the damaged fibres, the collagen fibres slide past one another, and breaks in the cross-links cause denaturation of the tissue. This also affects the non-collagenous matrix and vascular elements of the tendon leading to tendinosis (Paavola et al., 2002). If the strain is greater than 8%, rupture can occur (O'Brien, 1992) (Figure 2.6.).

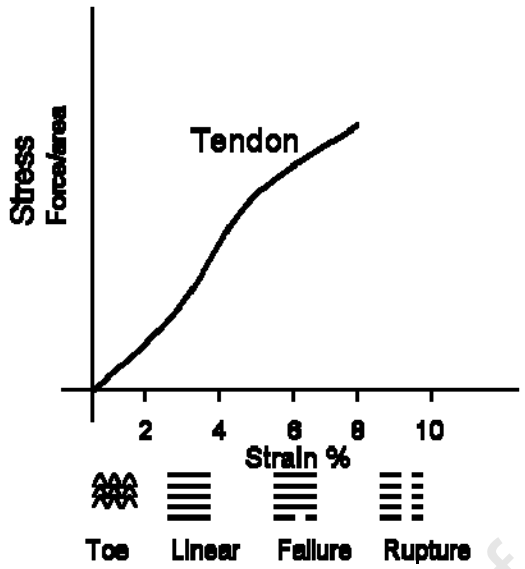

Figure 2.6. Stress/Strain curve of a tendon
Adapted from Novacheck TF., 1998 Running Injuries: A biomechanical approach. Instructional Course Lecture. 47: 397- 406.

It appears that there is a difference in tendon mechanical stress tolerance between males and females. This is probably related to the different anatomy, as the average tendon length is around 68mm in men and 60mm in women (Koike et al., 2004). Furthermore, the tendons in men have a larger cross sectional area than those in women, tendons in males have a higher maximum rupture force and stiffness (Thermann et al., 1995). Concerning the mechanical proprieties of the Achilles tendon, it is known that actin and myosin are present in the tenocytes. Furthermore, tendons have a good transmission of mechanical force from muscle to bone (Kader et al., 2002).

2.3.3.3 Biomechanics of the Achilles tendon during running

The biomechanics of the Achilles tendon during running has been studied using biomechanical models (Scott and Winter, 1990) or through direct *in vivo* measurements in the Achilles tendon (Komi et al., 1992).

The estimated peak load of the Achilles tendon at average running speed is around 6.1-8.2 times the body mass, and the tensile force can reach more than 3kN (Scott and Winter, 1990). However, peak forces can reach up to 9kN or 12.5 times the body mass at a running speed of 6m/s (Komi et al., 1992).

During a gait cycle, the Achilles tendon stretches during the first half of stance phase when it stores energy. It then recoils during the second half of the stance phase in a spring-like fashion and this returns 90% of the energy at the time of toe-off (Novacheck, 1998a). The peak ankle moment occurs at mid-stance and is generated by the muscle contraction of the triceps surae rather than through impact with the ground. It has been postulated that injuries of the Achilles tendon are caused by muscle forces during the midstance phase, rather than impact at heel contact. Therefore, the shock absorption capacity of the running shoe at heel strike may not play such an important role in this type of injury as has been postulated. However, the running shoe may have an important role in reducing the moment on the ankle at midstance through the reduction of ground reaction forces and by improving of stability (Novacheck, 1998a).

2.3.3.4 Summary: Biomechanics of Achilles Tendinopathy

It has been shown that if the Achilles tendon is stretched beyond 4% of its length, injury can occur. Ruptures can occur if the strain application is too frequent or if the magnitude of the strain exceeds 8%. During running, the load on the Achilles tendon can go from 6 to 12.5 times the body mass, depending on the running speed. It has been suggested that the mid stance is the critical phase of loading the Achilles tendon during running and this is when the Achilles tendon is probably more prone to injury due to the increase in the ankle moment. Therefore, it can be implied that

interventions (e.g. shoes, running technique, and orthotics) that may modify the ankle moment during running, may be appropriate to reduce the load on the tendon.

2.3.4 Definition and classification of Achilles tendon injuries

2.3.4.1 Definition of Achilles tendon injury

An overuse injury in a tendon can be defined as an injury that develops when the tendon is subjected to repeated strain until it cannot withstand further loading and damage occurs (McLauchlan and Handoll, 2001). At the molecular level, this is the point where the collagen cross-links begin to break. The term 'tendonitis' has been used to describe this condition and this implied that the pathology is as a result of inflammation. However, biopsies from patients with chronic Achilles tendon injury have not revealed inflammatory cells. Furthermore, studies have not found mediators of inflammation such as prostaglandin in a higher concentration in patients with Achilles tendon injury than in controls (Alfredson et al., 1999; Paavola et al., 2002).

2.3.4.2 Classification of Achilles tendon injuries

The classification of Achilles tendon injuries can therefore be confusing as there are several terms which have linked the pathology to inflammation including: tenosynovitis, tenovaginitis, peritendinitis, or paratenonitis (Schepsis et al., 2002). Furthermore, if pathologies coexist the distinction between groups of pathologies becomes even more unclear. Therefore, the classification as suggested by Puddu et al. (1976) which discern between peritendinitis; peritendinitis with tendinosis and tendinosis should be adopted:

1) Peritendinitis - This refers to inflammation of the paratenon without an inflammatory response of the tendon.

2) Peritendinitis with tendinosis - This refers to a second stage of injury in which the Achilles tendon and paratenon are both involved. The combination of paratendinitis with tendinosis may result in localized tenderness, typically 2 - 6 cm above the insertion, together with swelling and nodular deformity. Without the nodular

deformity, it is often difficult to distinguish between paratendinitis and paratendinitis with tendinosis.

3) tendinosis - This refers to an asymptomatic degenerative injury of the Achilles tendon without alteration of the paratenon. Furthermore, in Achilles tendinosis, there are no signs of inflammation (Jones et al., 1986). Tendinosis can be distinguished from partial rupture by a gradual increase in painful condition while partial rupture is associated with a sudden onset of pain (Alfredson and Lorentzon, 2000).

It has been recommended that the combination of pain in the tendon, swelling and performance impairment should be termed "tendinopathy", and this could include paratendinitis and tendinosis (Maffulli et al., 1998). For the purpose of this thesis, the terminology "Achilles tendinopathy" suggested by Mafulli et al. (2002) will be adopted.

Furthermore, Achilles tendinopathy condition has been sub-divided into insertional and non-insertional tendinopathy as they present a different aetiology and treatment (Clain and Baxter, 1992; Krishna and Maffulli, 2005):

1) Insertional Achilles tendinopathy is perhaps caused by a heel bump (Haglung deformity), which compresses the tendon against the shoe counter. It is characterized by an inflammation of the enthesis (the site where tendons and ligaments are attached to the bone).

2) Non-insertional Achilles tendinopathy, which is more common among athletes, is a degenerative disorder that may result from overuse of the tendon due to training errors, equipment or lower limb misalignments (Alfredson, 2003; varez-Nemegyei and Canoso, 2006). Typically, in non-insertional Achilles tendinopathy, soft tissue diagnostic ultrasound imaging reveals an abnormal tendon structure, nodular thickening and disrupted fibre orientation (Williams, 1986; Marti et al., 1988; Kvist, 1994). The observed morphological characteristics are changes in collagen fibre structure and arrangements, as well as an increase of interfibrillar glycosaminoglycans (Alfredson and Lorentzon, 2000).

Therefore, it can be concluded that Achilles tendinopathy, which is the injury investigated in this thesis, is a condition that includes peritendinitis and tendinosis. It can involve inflammation of the paratenon, but typically is a degenerative condition of the Achilles tendon that is associated with morphological changes such as nodular deformity.

2.3.5 Diagnosis of Achilles Tendinopathy

The diagnosis of Achilles tendinopathy is typically made by clinical assessment and then confirmed using diagnostic soft tissue ultrasound or magnetic resonance imaging (MRI) scanning. In the early stages, stiffness (usually in the early morning) is the only symptom, but as the condition progresses, pain develops. In addition, there can be associated decreases in range of motion, swelling and weakness during activity. Pain may occur during exercise and in some cases can even interfere with day to day activity (Kader et al., 2002). During training, pain is normally experienced at the beginning and at the end of a training session and sometimes discomfort occurs during training.

After a medical history has been obtained, the patient is examined in the standing and prone positions. On clinical examination, the tendon is tender to palpation, there may be an increased tissue temperature and reputations may be felt (Maffulli and Kader, 2002; Koike et al., 2004). The tenderness normally occurs in the mid portion of the tendon (1.5-7.0 cm proximal to the calcaneus) but in some cases, it can also extend proximally and distally (tendon and bone junction) (Alfredson and Lorentzon, 2000). In late stages of the condition, there can be a nodular appearance (Scioli, 1994; McCrory et al., 1999). Ultrasound and MRI are the common imagining methods that are used to confirm the clinical diagnosis of Achilles tendinopathy.

During ultrasound scan, the sensitive indicator of tendon substance abnormality is an increase in tendon thickness and echogenicity, which normally are seen in the medial third of the Achilles tendon (Koike et al., 2004). The ability of ultrasound of detecting abnormalities of the Achilles tendon is reported with a sensitivity of 80% and a specificity of 76% (Sell et al., 1996).

The MRI scan can depict the pathology of Achilles tendinopathy in great detail. However, the therapeutic guideline and the importance in clinical decision-making, based on MRI imaging, has not been clearly established (Kader et al., 2002). Patients with pain in the main body of Achilles tendon typically exhibit 1) thickened paratenon, 2) peritendinous fluid, 3) oedema of Kager's fat pad, 4) a thickened tendon in a fusiform shape, 5) focal or diffuse intratendinous intermediate or high signal, and 6) interrupted appearances of the tendon tissue (Kader et al., 2002).

2.3.6 Aetiology of Achilles tendinopathy

As previously mentioned there are several extrinsic and intrinsic factors that have been associated with running injuries such as Achilles tendinopathy (Kader et al., 2002). In this section, the evidence for each of the risk factors associated with Achilles tendinopathy will be reviewed. As in the section where general risk factors for running injuries were reviewed (Section 2.2 *A Review of Epidemiology of Running Injuries*), an evidence-based approach will be followed. More specifically, extrinsic factors for Achilles tendinopathy that will be reviewed include training errors, poor footwear and running on a hard running surface. Intrinsic factors for Achilles tendinopathy that will be reviewed are older age, gender, muscle strength, inflexibility, lower limb abnormalities, biomechanics and genetic predisposition.

2.3.6.1 Extrinsic risk factors for Achilles tendinopathy

2.3.6.1.1 Introduction

There are limited studies on extrinsic factors that may be associated with Achilles tendon injuries. The most frequently reported extrinsic factors for Achilles tendinopathy are training errors, poor footwear and running on a hard surface.

2.3.6.1.2 Training errors

Training errors have consistently been associated with the aetiology of several running injuries (Sallade and Koch, 1992; Johnston et al., 2003; Hreljac, 2005). In this

section, training errors are considered to be increased training distance and intensity or limited warm-up.

Increase training distance and intensity

In a case control study (McCrory et al., 1999), there was a tendency of runners with Achilles tendinopathy to have a higher weekly mileage (15% higher) than uninjured control runners. Additionally, injured runners tended to train at a faster running pace and for longer (more years) than controls. In a retrospective clinical report over a two year period, training errors were implicated in 75% of the cases with Achilles tendinopathy cases (Clement et al., 1984). The most common training errors that were identified included sudden increases in the training mileage, a severe competitive or training season, and sudden increases in training intensity (Clement et al., 1984).

Achilles tendinopathy is generally associated with overuse from repetitive loading (Archambault et al., 1995; Alfredson and Lorentzon, 2000). Although the effect of loading during exercise appears to be important for the development of tendon mechanical strength, it has also been suggested that during adaptation to a new load, there are periods of mechanical weakness, which may provoke injury (Archambault et al., 1995). The effects of repeated loading may have a negative effect if the loads are beyond the physiological limit of the tendon, or if they are too frequent, which does not leave time for repair and adaptation (Clement et al., 1984).

Achilles tendinopathy has also been observed among individuals who are not physically active (Alfredson and Lorentzon, 2000). In one study of 58 Achilles tendinopathy patients, it was documented that 31% of them had no direct association with sport or physical activity (Rolf and Movin, 1997). It has been suggested that physical activity may induce the symptoms rather than cause the injury (Alfredson and Lorentzon, 2000).

Therefore, it can be concluded that increases in training distance and training volume are associated with Achilles tendinopathy. However, the fact that Achilles tendinopathy can also be present in sedentary individuals might indicate that training just reveals the symptoms rather than cause the condition.

Limited warm-up

It has been hypothesized that limited warm-up could be a risk factor in developing Achilles tendinopathy (Milgrom et al., 2003; Barr and Harrast, 2005). The rationale behind this hypothesis is that with a decrease in temperature, there is a decrease in viscosity of the mucopolysacahrides, which act as a lubricant to the paratendinous structure. A reduction in viscosity would limit the gliding and smoothness of the tendon, which could then induce injury.

In one study among military recruits it was found that there was a significantly higher incidence of Achilles paratendinitis in winter compared with summer (Milgrom et al., 2003). The researchers have suggested that this may be related to a decrease in temperature of the Achilles paratenon, resulting in a reduction in the viscosity of the mucopolysaccharides (as mentioned before).

In summary, the results of these studies on runners with Achilles tendinopathy are aligned with studies from multi-injuries (Yeung and Yeung, 2001a; Hreljac, 2005), which show that increased training distance and intensity are an associated risk factor. Furthermore, warming-up may play a role in the risk of developing this injury.

2.3.6.1.3 Footwear

The use of incorrect running shoes has been postulated as one of the risk factors that may be associated with running injuries, including Achilles tendinopathy (McKenzie et al., 1985; Barnes and Smith, 1994; Lake, 2000). According to McKenzie et al. (1987), from the early 1970's to the late 1980's, the incidence of Achilles tendon injuries has decreased while the incidence of knee injuries has increased. It has been suggested that this may be due to the introduction of heel wedging above 10mm and, therefore, an increase on shoe stability.

The only retrospective study that investigated the effect of the shoe on Achilles injury found that 10% of the Achilles injuries were related to poor footwear (Clement et al., 1984), however, the definition of a poor footwear was not presented. They suggested that heel wedges should be maintained between 12 - 15 mm, otherwise there will be

an increase in strain over the Achilles tendon (Smart et al., 1980; Clement et al., 1984).

In summary, there is limited evidence of the importance of footwear on the development of running injuries, therefore, prospective studies, which look to particular characteristics of running shoes (e.g. age, midsole hardness, motion control) and specifically to the Achilles tendon injury population should be developed.

2.3.6.1.4 Running surface

Running on a hard surface has been commonly associated with running injuries in general (Macera et al., 1989; Messier et al., 1991). The only study that has investigated this aspect in Achilles tendon injured runners found that these runners tend to run over dirt surfaces and less on asphalt than uninjured runners (McCrory et al., 1999). It has been postulated that running on crowned or uneven road and slippery terrain can generate overpronation, which may predispose the runner to Achilles tendinopathy (Clarke et al., 1984; McCrory et al., 1999). It can be concluded that running on uneven surfaces might predispose runners to Achilles tendinopathy injury but further studies are necessary to examine this aspect.

2.3.6.1.5 Summary: Extrinsic risk factors for Achilles tendinopathy – an evidence-based approach

From the existing published data and from the support provided through the evidence-based medicine (EBM) criteria (Obremskey et al., 2005) illustrated in Table 2.5, it appears that training errors such as high training mileage and limited warm-up may be important extrinsic factors in the aetiology of Achilles tendinopathy. However, they are probably not the cause of injury as this type of injury is commonly seen in sedentary participants (Rolf and Movin, 1997)

Table 2.5. Extrinsic risk factors associated to Achilles tendinopathy using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Training errors	**A. Increase training distance**	
	Positive association:	
	Retrospective study: (Clement et al., 1984)	II
	Case control: (McCrory et al., 1999)	III
	B. Increase training intensity	
	Positive association:	
	Retrospective study: (Clement et al., 1984)	II
	Case control: (McCrory et al., 1999)	III
	C. Limited warm-up	
	Positive association:	
	Prospective study: (Milgrom et al., 2003)	I
	Expert opinion: (Barr and Harrast, 2005)	V
Footwear	**Positive association:**	
	Retrospective study: (Clement et al., 1984)	II
	Systematic review of level II: (Smart et al., 1980)	II
	Expert opinion: (McKenzie et al., 1985)	V
Running surface	**Negative association:**	
	Case control: (McCrory et al., 1999)	III

2.3.6.2 Intrinsic risk factors for Achilles tendinopathy

2.3.6.2.1 Introduction

Intrinsic factors that may be associated with Achilles tendon injuries have been examined more extensively when compared to the extrinsic factors. The most frequently intrinsic risk factors that are reported for Achilles tendinopathy are older age, gender, muscle weakness and imbalance, inflexibility, lower limb abnormalities, biomechanical parameters, and an inherent genetic predisposition.

2.3.6.2.2 Age

Achilles tendinopathy is characterized by degenerative changes in the fibre structure, therefore, it has been suggested that age will influence the risk of developing this injury (Astrom and Rausing, 1995).

According to a recent review study, Achilles tendinopathy is most frequently seen in runners aged 35-45 years (Alfredson and Lorentzon, 2000). In one study of 109 runners who suffered from Achilles tendinopathy over a period of two years it was shown that the injured runners had an average age of 39 years. These values tend to be higher when compared with runners who develop other running injuries that are seen in a running clinic (1650 injured runners), where the average age was 30 years old (Clement et al., 1984). Moreover, in a prospective study conducted over three years, Achilles tendon disorders were significantly more common among elderly athletes (over 60 years old) than among young athletes (21 to 25 years old) (20% vs. 5%, respectively) (Kannus et al., 1989). Thus, there is strong evidence to lend support to the notion that individuals in older ages are more predisposed to develop Achilles tendinopathy.

2.3.6.2.3 Gender

Male gender has been associated with increased risk of Achilles tendinopathy (Clement et al., 1984; Astrom and Rausing, 1995). In one case control study it was found that increasing age and male gender were associated with more pronounced histopathologic changes in the tendons of patients with chronic Achilles tendinopathy (Astrom and Rausing, 1995). In one retrospective study of 109 runners with Achilles tendinopathy, it was noted that 78% of the runners were male (Clement et al., 1984).

In summary there is some, but limited, evidence to suggest that males, despite having larger cross sectional area and longer Achilles tendons as aforementioned (Thermann et al., 1995; Koike et al., 2004), are at increased risk of developing Achilles tendinopathy, compared with females.

2.3.6.2.4 Muscle weakness

It has been postulated that gastrosoleus weakness can be a risk associated factor, as weakness or fatigue may limit the energy-absorbing capacity of the muscle and increase the overload on the tendon (Paavola et al., 2002). Others suggest that muscle imbalances may also be an aetiological factor in running injuries (Alfredson et al., 1998b).

In a prospective study with military recruits in which running was part of their training program, found that a reduction in plantarflexion strength was a predictor of Achilles tendon overuse injury (Mahieu et al., 2006). Similary, muscle strength was a discriminator factor in a case-control study with Achilles tendinopathy runners, as injured runners presented a significantly higher dorsiflexion peak torque but a lower plantarflexion peak torque at 60°/s and 180°/s (McCrory et al., 1999). Mc Crory et al. (1999) also found similar muscle strength of the injured and non-injured leg, which was interpreted as the strength deficiency that was present before the manifestation of the injury.

It was found that patients who were recovering from surgical treatment for Achilles tendinopathy showed a reduction in concentric muscle strength on the injured side compared with the non-injured side even 52 weeks after surgery; however, eccentric torque was similar after this period (Alfredson et al., 1998b). The researchers concluded that concentric and eccentric calf muscle training can be important in the prevention of Achilles tendinopathy. However, they also highlighted that this cannot be interpreted as a cause-effect, as changes in the tendon due to tendinosis and associated pain could be the cause of reduced muscle strength (Alfredson et al., 1998b; Alfredson and Lorentzon, 2000).

In summary, there is some, but limited, evidence to suggest that muscle weakness, especially in the calf muscles, may be a risk factor for Achilles tendon injuries.

2.3.6.2.5 Inflexibility

There is not an established agreement whether the reduction in muscle flexibility is an associated aetiological factor for running injuries (Warren and Jones, 1987; van Mechelen et al., 1992; Hreljac et al., 2000). However, in Achilles tendinopathy, there seems to be a better correlation of the decrease in flexibility and the pathology (Clement et al., 1984; Hess et al., 1989; Kvist, 1994; Alfredson and Lorentzon, 2000).

It has been speculated that the relation between Achilles tendon injury and flexibility is perhaps related to age; as reported before age is a strong aetiological factor of Achilles tendinopathy (Kannus et al., 1989), and as reduction in muscle flexibility is commonly seen in middle-aged individuals (Kvist, 1994).

In a number of reviews, it is frequently recommended that flexibility training of the calf muscles is important in the treatment of patients with Achilles tendinopathy (Hess et al., 1989; Kvist, 1994; Alfredson and Lorentzon, 2000). In a case-control study involving 31 runners with Achilles tendon injury and 58 controls, it was shown that injured runners were less likely to incorporate stretching into their daily routine (McCrory et al., 1999). However, the quality of stretching was not addressed and both groups indicated that they do not stretch regularly.

Although, reduced range of motion in the ankle joint is commonly seen as a predictor of Achilles tendinopathy, a prospective study with army recruits found the opposite as increase in dorsiflexion excursion was associated with a greater risk of developping Achilles tendinopathy (Mahieu et al., 2006). In contrast, in a retrospective study, poor flexibility of the gastrocnemius and soleus unit as measured by decreased range of movement in plantar and dorsiflexion was associated with risk of Achilles tendon injury (Clement et al., 1984). In this study it was suggested that the reduced ankle flexibility may be associated with increased knee flexion or increase pronation during walking and running, and that these increases in movement of the knee and foot may increase the risk of injury. Therefore, weakness of the gastrocnemius and soleus muscles during eccentric actions, combined with reduced flexibility of these muscles, could reduce dorsiflexion in the stance phase, which may increase the risk of Achilles tendon injury, however this requires further investigation.

2.3.6.2.6 Lower limb abnormalities

It has been suggested that two thirds of Achilles tendon disorders are due to anatomical misalignments or biomechanical problems (Kvist, 1994). Misalignment of the lower limb such as forefoot varus, limited passive subtalar and ankle joint mobility were found in 60% of Achilles tendinopathy patients (Kvist, 1994). Another anatomical factor associated with the injury in a case control study was cavus feet, as injured runners presented a significantly lower arch index (a higher arch) than uninjured runners (McCrory et al., 1999). Furthermore, a retrospective study showed that mild to severe foot varus alignment was observed in 87% of the patients with Achilles tendinopathy (Clement et al., 1984). However, a prospective study did not find any association between static measurements and lower limb injuries including ankle injury (Lun et al., 2004).

It was also been suggested that some lower limb anthropometrical measurements may predispose runners to injury by increasing pronation (Clement et al., 1984; McCrory et al., 1999) This hypothesis will be reviewed in the next section 2.3.6.2.7. *Biomechanical factors*.

In summary, there are a limited number of studies in the literature that have investigated the effect of lower limb abnormalities and Achilles tendinopathy (Clement et al., 1984; Kvist, 1994; McCrory et al., 1999) and some of them did not present a control population (Clement et al., 1984; Kvist, 1994). Therefore, it cannot yet be concluded that there is a high association of this variable and Achilles tendinopathy as previously stated (Kvist, 1994).

2.3.6.2.7 Biomechanical factors

It has been suggested that overpronation is one of the most important aetiological factors in Achilles tendinopathy (Clancy, Jr. et al., 1976; Clement et al., 1984; Kvist, 1994; Jones, 1998; Paavola et al., 2002). It has been suggested that pronation promotes an internal tibia rotation, which dislocates the Achilles tendon medially and may produces a whipping action on the Achilles tendon and this increased force can causes microtears in the medial part of the tendon. It has also been speculated that the

asynchrony between knee extension and pronation may produce conflicting rotational forces, which may wring the vessels in the tendon and peritendon causing vascular impairment and possible degeneration (Clement et al., 1984). However, the epidemiological studies that have investigated the biomechanics causes of Achilles tendinopathy are still limited.

Several studies have attempted to related some static measurements to overpronation to Achilles tendon injury (Clancy, Jr. et al., 1976; Clement et al., 1984; Kvist, 1994; Jones, 1998; Paavola et al., 2002). An association between pronation and Achilles tendinopathy was found in a case report of five runners (Clancy, Jr. et al., 1976). Review studies have also claimed that excessive pronation of the forefoot which induces pronation is the major aetiological factor of this injury (Kvist, 1994; Jones, 1998; Paavola et al., 2002). Clement et al. (1984) have suggested in a retrospective study that pronation is intimately related to varus alignment of the forefoot, heel and distal shaft of the tibia. They have also speculated that 56% of the Achilles tendon patients seen during the study have functional overpronation due to severe varus foot alignment.

Although there are some studies that relate static measurements with overpronation and, therefore, injury, as far as is known, there is only one study that investigated overpronation during running in runners with Achilles tendinopathy (McCrory et al., 1999). This study, which used 2D video analysis, found that maximum pronation, time to maximum pronation and maximum pronation velocity were significantly higher on the Achilles tendinopathy group than the control group (McCrory et al., 1999). Furthermore, calcaneus touchdown angle was significantly lower among the injured group. These results indicate that the Achilles tendinopathy group had a more inverted foot at touchdown and a higher pronation and pronation velocity during stance phase. The same study was also the only one to investigate running kinetics in runners with Achilles tendinopathy; however, no significant differences were found when compared with the uninjured group (McCrory et al., 1999).

In summary functional overpronation has been associated with Achilles tendinopathy, but there is only one study, which investigated the biomechanics of Achilles

tendinopathy runners during running (McCrory et al., 1999). Therefore, it cannot yet be established that running biomechanics parameters are associated with this injury. Furthermore, intra-participant and inter-participant variability of biomechanical parameters as possible intrinsic risk factors for Achilles tendinopathy has not been studied. These parameters as possible intrinsic risk factors for Achilles tendinopathy will be the focus of one experimental study presented in this thesis.

2.3.6.2.8 Genetic predisposition

According to recent investigations, Achilles tendinopathy may have a genetic predisposition (Mokone et al., 2006; September et al., 2008). More precisely, the gene alpha 1 type V collagen (COL5A1), which encodes a tendon protein, was found to be higher in the Achilles tendinopathy population compared to a control population in South Africa (Mokone et al., 2006). The same findings were observed in an Australian population (September et al., 2008), thus indicating that this gene may predispose individuals to develop Achilles tendinopathy.

2.3.6.2.9 Summary: Intrinsic risk factors for Achilles tendinopathy – an evidence based approach

In conclusion male runners, older than 35 years old, with weak and restricted flexibility of the gastrocnemeius and genetic predisposition may be considered possible risk factors to Achilles tendinopathy. However, there is a need to further investigate the association between lower limb alignment and biomechanics in the aetiology of this injury.

Table 2.6. Intrinsic risk factors associated to Achilles tendinopathy using the evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Age	**Positive association:**	
	Prospective study: (Kannus et al., 1989)	I
	Retrospective study: (Clement et al., 1984)	II
	Systematic review level I-III studies: (Alfredson and Lorentzon, 2000)	III
Gender	**Positive association (Male):**	
	Retrospective study: (Clement et al., 1984)	II
	Case control: (Astrom and Rausing, 1995)	III
Muscle weakness	**A. Muscle weakness**	
	Positive association	
	Prospective study: (Alfredson et al., 1998b; Mahieu et al., 2006)	I
	Case control: (McCrory et al., 1999)	III
	Expert opinion: (Paavola et al., 2002)	V
Inflexibility	**Positive association**	
	Prospective study: (Mahieu et al., 2006)	I
	Retrospective study (Clement et al., 1984)	II
	Systematic review level I-III studies: (Hess et al., 1989; Kvist, 1994; Alfredson and Lorentzon, 2000)	III
Lower limb abnormalities	**Positive association**	
	Prospective study: (Lun et al., 2004)	I
	Retrospective study: (Clement et al., 1984)	II
	Case study: (McCrory et al., 1999)	
	Systematic review level I-III studies: (Kvist, 1994)	III

Table 2.6 continued. Intrinsic risk factors associated to Achilles tendinopathy using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Biomechanical factors	**A. Functional overpronation**	
	Positive association:	
	Retrospective study: (Clement et al., 1984; Kvist, 1994)	II
	Systematic review of level II (Jones, 1998; Paavola et al., 2002)	II
	Systematic review level I-III studies: (Kvist, 1994)	III
	Case series: (Clancy, Jr. et al., 1976)	IV
	B. Dynamic overpronation	
	Positive association:	
	Case control: (McCrory et al., 1999)	III
	C. Kinetic parameters	
	No association:	
	Case control: (McCrory et al., 1999)	III
Genetic predisposition	**Positive association:** Case control: (Mokone et al., 2006; September et al., 2008)	III

2.3.6.3 Summary: Risk Factors for Achilles tendinopathy

Extrinsic and intrinsic risk factors have been associated with the aetiology of Achilles tendinopathy. There is strong evidence that extrinsic risk factors such as training errors, more specifically increased training distance and limited warm-up are associated with the injury.

There is also some evidence to suggest that Achilles tendinopathy is more common in older, male runners. Other aetiological factors, which seem to be associated with injury are gastrosoleus weakness and imbalance, and poor flexibility (Table 2.6). Overpronation has been frequently listed as an important injury risk factor. However, most of the evidence comes from clinical studies and there is a lack of experimental studies presented in the literature. Similarly, reduced variability of biomechanical parameters as a possible risk factor for Achilles tendinopathy has not been studied.

Finally, recent studies have provided some sound evidence of genetic predisposition in this injury.

2.3.7 Management of Achilles tendinopathy

The main focus of this thesis is on the identification of factors associated with Achilles tendon injury rather than the management of the injury. Therefore, a detailed discussion of the management of Achilles tendinopathy is beyond the scope of this review and this thesis. Therefore, only a brief discussion on the general approach to management of Achilles tendinopathy is presented.

It is well established that in the initial stages of the injury, conservative (non-operative) treatment is the most effective treatment and is successful in most cases (Clement et al., 1984; Hess et al., 1989; Alfredson and Lorentzon, 2000). However, in some refractory cases, surgery is indicated (Alfredson and Lorentzon, 2000). The modalities of non-operative treatment for Achilles tendinosis will be briefly discussed.

According to a recent Cochrane review, there is insufficient evidence to determine which method is the most appropriate to treat acute or chronic Achilles tendinopathy (McLauchlan and Handoll, 2001). Therefore, the conventional methods to treat Achilles tendinopathy will be briefly discussed in no particular order.

2.3.7.1 Correction of biomechanical abnormalities

Corrective orthotics have been prescribed frequently for the treatment and prevention of running injuries in general (MacLean, 2001). Similarly, orthotics are often prescribed to treat Achilles tendinopathy. The main rationale for this treatment modality is the correction of excessive pronation, forefoot varus or rearfoot varus. Heel lifts to reduce tension of the calf muscle and the Achilles tendon have also been used. However, it has been shown that a heel pad is not effective in some patients with Achilles tendinopathy (Lowdon et al., 1984).

2.3.7.2 Training adjustments

As mentioned in a previous section (2.2.3 *Grading of the severity of running injuries*), overuse injuries can be classified into grades 1-4. Therefore, the training routine must adjusted according to the grade of injury. If the pain from Achilles tendinopathy restricts performance (Grade 3 or 4), a complete rest or modified training with non-weight bearing exercises (e.g. cycling, swimming), is frequently recommended (Alfredson and Lorentzon, 2000).

2.3.7.3 Stretching exercise

Calf muscle stretching is commonly suggested as an essential treatment method for Achilles tendinopathy (Alfredson and Lorentzon, 2000). It has been shown that stretching treatment improves muscle compliance, and this may cause a reduction in injury risk (Rosenbaum and Hennig, 1995). These results of treatment using stretching appear to be more significant when warm-up is incorporated in association with stretching as this can promote an additional improvement in muscle force (Rosenbaum and Hennig, 1995).

2.3.7.4 Eccentric training for the calf muscles

There is good evidence to suggest that eccentric calf exercises are an effective method of treatment for Achilles tendinopathy. In some cases, eccentric calf exercises are even more beneficial than conventional treatment, which includes rest, medication, and orthotics (Stanish et al., 1986; Alfredson et al., 1998a).

The precise reason of the positive response to this type of treatment is still unknown but it can be related to an increase in tensile strength or lengthening of the muscle-tendon unit, which can reduce the strain on the ankle joint (Alfredson and Lorentzon, 2000). Another theory is based on the energy supply of the tendon tissue. It is known that metabolism within the tendon changes from aerobic to anaerobic (Hess et al., 1989). Therefore, it has been suggested that the tendon must adapt to the more anaerobic demand (Alfredson and Lorentzon, 2000). Eccentric exercises might result in transient tendon ischemia, and therefore promote physiological anaerobic

adaptation. This mechanism has been suggested as one reason why eccentric calf exercise is effective in the treatment of Achilles tendinopathy (Alfredson et al., 1998a; Alfredson and Lorentzon, 2000).

2.3.7.5 Medication

The use of corticosteroid injections in and around the Achilles tendon is controversial. It has been reported in several studies, that there may be an increase in partial or complete ruptures of the Achilles tendon following corticosteroid injection (Ljungqvist, 1967; Leadbetter, 1995). Furthermore, because there are no signs of inflammation on Achilles tendinopathy, the use of corticosteroids does not seem logical. Non-steroidal anti-inflammatory drugs (NSAIDs) have also been frequently used. However, once again there is no support from the literature on the efficacy of this treatment (Ljungqvist, 1967). Therefore, more recently it has been suggested that for the treatment of pain in Achilles tendinopathy other medication with fewer adverse effects can be used (Alfredson and Lorentzon, 2000).

2.2.7.6 Surgery

In cases where non-operative treatment is not successful, surgery may be recommended. Surgery is required in 25% of the cases and can increase according to patient age, duration of symptoms and occurrence of tendinopathic changes (Kvist, 1994). A detailed discussion of possible surgical procedures that may be used in the treatment of Achilles tendinopathy is beyond the scope of this review.

2.2.7.7 Summary: Management of Achilles tendinopathy

There are a number of treatment options for Achilles tendinopathy including rest, ice, non-steroidal anti-inflammatory (NSAIDs), local corticosteroids injections, inserts and a specific calf muscle eccentric rehabilitation program. It appears that there is no single most appropriate method of treatment for Achilles tendinopathy (McLauchlan and Handoll, 2001). Therefore, most clinicians would use a combination of various therapies in the treatment of this injury.

2.4 A Review of the Biomechanics of Running Shoes

2.4.1 Introduction

One can assume that the role of running shoes is related to injury prevention and performance improvement. In relation to performance, some studies have investigated the role of running shoe on the improvement of running economy (Anderson, 1996). According to the review, decrease in shoe mass and an increase in cushioning may improve running performance.

However, the role of running shoes in injury prevention is the area that intrigues most of the researchers (McKenzie et al., 1985; Subotnick, 1985; Frederick, 1986; Lake, 2000). The rationale of the role of the running shoe in injury prevention is related to the accepted characteristic of running shoes to reduce impact force and control pronation. However, as it will be further explored, there is in fact very limited evidence and conflicted studies in this subject. To understand the role of running shoes in preventing injuries, there is a need to understand the biomechanics of running with shoes and the biomechanical effect of different shoe proprieties.

2.4.2 A brief historical development of running shoe testing

It was only in the 1950s that athletic shoe manufacturers started to test shoes under a more controlled conditions. However, it was not until the 1970s that biomechanical tests started to have an influence on the design of footwear (Bates, 1985). In the earlier tests, the focus was on shock absorption, which was considered to be the most important characteristic of a running shoe, as impact forces can reach up to 3 times the body mass during running.

In the later 1970s, some clinical studies identified overpronation as a possible risk factor for running injury (James et al., 1978; Bates et al., 1979). A few years later, the first studies were published in which altered shoe properties were incorporated to control pronation (Clarke et al., 1983b; Nigg and Segesser, 1986; Nigg and Morlock, 1987; Nigg et al., 1987). More recently, the concepts of impact force and pronation

have been challenged by a new theory (Nigg, 2001; Nigg and Wakeling, 2001). Nigg (2001) has suggested that impact forces are input signals that produce muscle tuning in order to minimize soft tissue vibration and to reduce joint and tendon loading. Nigg (2001) suggests that soft tissues packages, which are associated with the major muscle groups (triceps surae, hamstring and quadriceps), are close to the input frequency of ground reaction impact force (5 to 65 Hz) and these might cause resonance phenomena which can increase muscle vibration and joint loading. Hence, it was proposed that the muscle should be tuned before contact to minimize vibration (Nigg, 2001). The muscle tuning would affect joint stiffness and joint geometry including pronation along with other kinematics adaptations. This hypothesis is summarized as follows:

> *"The proposed solution suggests that the locomotor system uses a similar strategy in both situations "impact" and "movement control"….To deal with impact forces the muscles are pre-tuned to possibly minimize soft tissue vibration. To deal with the shoe, inserts and orthotics, the muscles are activated (if necessary) to provide a constant joint movement pattern. This strategy affects muscle activation during contact…The characteristic of individual subjects with respect to resonance frequencies of soft tissue packages and preferred joint movement paths are different. The subject specific reaction to shoes inserts and orthotics are experimentally measured."*
> Nigg (2001, p.8)

More recently, studies have focused their attention on the effect of running shoes on muscle activity, rather than purely on the kinetic and kinematics variables (Nigg and Liu, 1999; Wakeling et al., 2001; Wakeling et al., 2002b; Nigg et al., 2003; von Tscharner et al., 2003; Nigg et al., 2006a). Furthermore, the importance of variability in biomechanical parameters and how variability may be influenced by running shoes has become more important (Hamill et al., 1999; Kurz and Stergiou, 2003; Kurz et al., 2003).

2.4.3 Effects of running shoes on biomechanics

2.4.3.1. Running in shod compared to barefoot condition

In order to understand the effect of running shoes on biomechanics, it is necessary to initially compare running in shod condition to barefoot running. The studies analysed here define shod as the condition where running footwear in worn.

There is some evidence to suggest that vertical loading rate is significantly lower in shod than barefoot condition (Dickinson et al., 1985; De et al., 1994; De Wit et al., 2000). Other studies however, have also found a significantly lower vertical impact force peak in barefoot compared with shod condition (Divert et al., 2005a; Divert et al., 2005b).

The effect of running shoes on kinematics has also been studied. It has been shown that heel strike occurs in a more dorsiflexed position in shod than barefoot running (De Wit et al., 2000; Bishop et al., 2006). It has been suggested that the flat foot position is adopted by barefoot running is a strategy to limit the pressure underneath the heel (De Wit et al., 2000). Additionally, the knee is in a more flexed position at initial contact throughout midstance in the shod condition (De Wit et al., 2000).

Studies in which frontal plane kinematics has been studied have shown that running with shoes increases ankle inversion at touchdown and an increase total eversion and eversion velocity (Stacoff et al., 1991). Therefore, tibial rotation may decrease in the barefoot condition, and this may lead to fewer running injuries. However, when kinematics have been studied by inserting intracortical bone pins with reflective markers, it was found that differences in ankle eversion between barefoot and shod condition were small and unsystematic (Stacoff et al., 2000). The authors suggested that previous studies described the movement of the shoe or the skin rather than the movement of underlying bone. However, it should be noted that the five participants of this study were running under the effect of local anaesthetic, and this could affect the sensory feedback on the sole of the foot.

There is also an increase in stride length and a reduction in stride frequency in shod running resulting in lower leg stiffness (De Wit et al., 2000). This was supported by the finding of another study, in which found that vertical and leg stiffness was significantly lower in shod compared with barefoot running (Divert et al., 2005a).

Muscle activity in shod versus barefoot running has also been studied. Muscle activity of the tibialis anterior (TA) was shown to occur significantly later after heel strike in the shod condition. It was also observed that the intensity of TA EMG activity before heel strike increased, when compared with the after heel strike when wearing shoes. The authors concluded that EMG activity of the tibialis anterior adjusts to exterior conditions (von Tscharner et al., 2003).

Thus, running in a shod condition is associated with significant kinetic, kinematics, temporal distance and muscle activity adjustments. Finally, as mentioned previously (section 2.2.4.2.7 *Variability in biomechanical parameters*), there is an increase on stride-to-stride variability on kinematic parameters when participants run on barefoot compared to shod condition (Kurz and Stergiou, 2003).

2.4.3.2 Effect of shoe proprieties on running biomechanics.

Differences in shoe properties have been investigated for more than twenty years (Clarke et al., 1983b; Frederick, 1986; Milani et al., 1997; Nigg and Liu, 1999; Wakeling et al., 2002b). Nigg and Segesser (1992), have described a criteria for sports shoe construction. According to these authors, a sport shoe should limit the impact force during landing, support the foot during the stance phase and guide the foot during the final phase of ground contact. In this section different shoe proprieties will be reviewed including midsole hardness, heel flare, heel height edge and format, comfort and movement variability.

Midsole hardness

One of the shoe properties, which has consistently been investigated is the midsole hardness, as impact forces have been associated with running injuries (Hreljac et al., 2000; Hreljac, 2004). It has been suggested that shoes should reduce impact force in

the early part of stance phase for 30 to 50 ms when forces are higher (Cavanagh and Lafortune, 1980; Nigg et al., 1987).

One of the early studies (Clarke et al., 1983a) to compare the effect of midsole hardness on vertical impact forces found that the time to reach vertical impact force was significantly longer on soft shoes than hard shoes; however, no statistical differences were found on the magnitude of vertical impact force. These results were confirmed by a later study, in which the magnitude of vertical impact force peak was not altered by shoes with different hardness, but the time of vertical impact forces was affected (Snel et al., 1985). The researchers also did not find any relationship between the results of the material tests and the test with human participants on impact forces. They concluded that the material test is not a good method to compare running shoes and there should be neuromuscular adjustment to adapt the individual to different midsole stiffness (Snel et al., 1985).

There appear to be some kinematic adaptations in the knee and ankle joints, which regulate the impact forces (Nigg et al., 1987; Hardin et al., 2004). As studies which compared shoe with different hardness have shown, impact forces may be adjusted by an increase in pronation or pronation velocity (Clarke et al., 1983b; Nigg et al., 1987; Luethi et al., 1987) or by an increase in ankle dorsiflexion velocity (Hardin et al., 2004). These kinematics adjustments have suggested the existence of a mechanism to regulate impact force magnitude during running (Hardin et al., 2004). However, when kinematics adaptations are excluded, as in a study where an impact test via a pendulum on the shod heel was used, impact forces were significantly different between a hard and a soft midsole (Aerts and De, 1993).

Furthermore, there is a good correlation between biomechanical variables and perception of impact scores, using a categorical rating to judge impact force (Milani et al., 1997). It therefore appears that there is a neurosensory system that is capable of differentiating between different impacts, which could help the runner to alter running style (Milani et al., 1997). Neurosensory feedback on the sole of the foot appears to be an important mechanism to control gait movement (Chen et al., 1995; Nurse and Nigg, 2001). In studies where sensory input was altered through specially made socks with sand (Chen et al., 1995), or by applying ice (Nurse and Nigg, 2001), this resulted

in an alteration of the pressure distribution on the sole of the foot. Furthermore, ice intervention and orthotics also resulted in significant changes in muscle activity during walking and running cycles respectively (Nurse and Nigg, 2001; Mundermann et al., 2006).

Several studies have also shown that muscle activity may be affected by differences in shoe hardness (Komi et al., 1987; Wright et al., 1998; Wakeling et al., 2002b; Nigg et al., 2003). In one of these studies, a three dimensional musculoskeletal model of the lower extremity was created to simulate impact forces in two different shoe hardness conditions (Wright et al., 1998). Similary to other studies (Nigg et al., 1987; Hardin et al., 2004), it was found that vertical impact force peak does not change according to different shoe hardness, but vertical loading rate and rate of knee flexion were higher in the soft shoe condition. However, this study have found that there was not a consistent effect on muscle force according to different shoe conditions, as the tibialis anterior force was higher in the hard shoe condition, while peroneus force was higher with the soft shoe condition. It was concluded that changes in external forces do not correspond to changes in internal forces (Wright et al., 1998).

Heel flare

A second parameter in shoe construction that requires discussion is the heel flare. The heel flare is the lateral extension of the midsole and is illustrated in Figure 2.7. It has been postulated that running shoes, which have a prominent heel flare, could increase the lever about the subtalar joint and therefore increase ankle eversion (Nigg, 1986; Edington et al., 1990). Studies have shown that initial eversion and maximum eversion velocity are amplified with the increase of the heel flare (Nigg and Morlock, 1987; Kalin et al., 1988b). However, another study it was shown that a decrease in the heel flare resulted in a significant increase in maximal pronation (at midstance) and total rearfoot movement, and this can vary according to midsole hardness (Clarke et al., 1983b). Thus, by way of conclusion, increase in heel flare may affect the frontal plane ankle kinematics at initial contact but not at midstance.

However, in a study (Stacoff et al., 2001) using bone pin markers it was found that the increase in heel flare does not increase foot eversion velocity, or tibial rotation

velocity as previously postulated. This result corroborates the findings of the same researchers discussed earlier on barefoot and shod condition (Stacoff et al., 2000), where kinematic results using skin markers are not reproduced when bone makers are used.

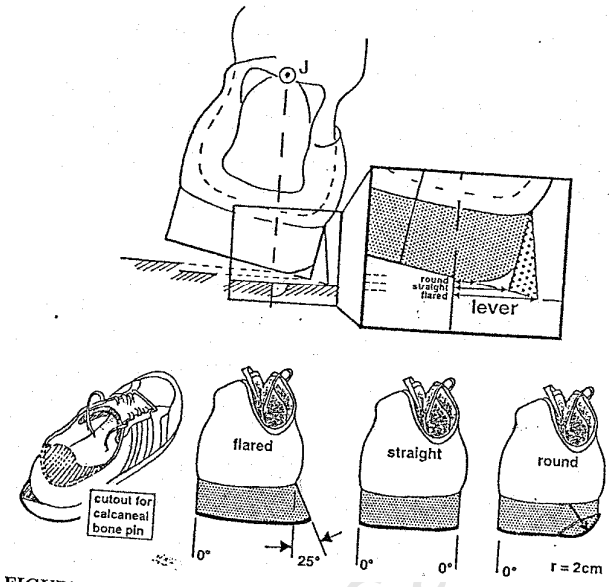

Figure 2.7. Running shoes with different flare
(MSSE. Stacoff et al, 2001, 23 (2), 311-319. Figure 3)

Heel height edge and format

Other alterations in shoe construction include changes in the edge of the heel height in order to turn the shoe as neutral, varus and valgus (O'Connor and Hamill, 2004). In one study, the valgus shoe condition was associated with a significantly increased maximum eversion, inversion moment and total negative work than the other two shoe conditions. In another study, Nigg (2004) has analysed the effect of rounded heel sole, regarded as an "unstable shoe" on kinetics, kinematics and EMG activity. In a standing condition, this shoe increased the centre of pressure excursion and EMG activity of the tibialis anterior. During walking, the rounded heel show caused an increase in dorsiflexion during the first half of the stance phase (Nigg, 2004).

As mentioned previously, Achilles tendon injury may be associated with an inadequate heel wedging in running shoes (Smart et al., 1980; Clement et al., 1984).

Also, heel lift has been suggested as a treatment for runners suffering with Achilles tendinopathy (Krissoff and Ferris, 1979), as this is expected to decrease the strain and forces on the Achilles tendon.

To understand the effect of heel lift on Achilles loading, ankle moment was compared between different heel heights during running (Reinschmidt and Nigg, 1995). It was established that ankle moment, which can be calculated through inverse dynamic, can be used to estimate Achilles tendon loading (Fukashiro et al., 1993). The magnitude and time of the initial dorsiflexion moment varied significantly between different heel heights. However, magnitude and time of maximum plantarflexion moment during midstance were not altered. Reinschmidt and Nigg (1995) concluded that heel lift does not alter Achilles loading. Furthermore, according to another study the increase of heel lift appears to have no effect on maximum pronation and total rearfoot movement according to a 2D kinematic analysis (Clarke et al., 1983b).

Comfort

A very important element in the design of a sport shoe is comfort, although the perception of comfort is variable according to the participant. In some earlier studies comfort was measured using a modified Borg scale (Miller et al., 2000), but more recently a study has validated the use of a visual analog scale (VAS) as a reliable method to measure comfort (Mundermann et al., 2002).

Mundermann et al. (2003) stated that 35 % of differences in comfort can be explained by the changes in 15 kinetic, kinematic and EMG variables. Therefore, comfort can be related to a change in running biomechanics. In support of these findings, another study has shown that shoe comfort decreases from walking to running (Miller et al., 2000).

As mentioned previously, midsole hardness does not influence vertical impact forces (Clarke et al., 1983a; Snel et al., 1985; Nigg et al., 1987; Hardin et al., 2004). However, midsole hardness can influence participant comfort (Milani et al., 1997; Lake and Lafortune, 1998). Comfort was perceived as less when military recruits had to use no inserts or hard inserts rather than soft inserts (Mundermann et al., 2001).

Nevertheless, there was participant variability in the perception of comfort. Foot arch height, foot and leg alignment and foot sensitivity were participant characteristics which were more related to comfort perception. In support of these finding other studies have related shoe fit (Hawes et al., 1994) and lower limb alignment (Nigg et al., 1999) to comfort perception. Finally, in another study shoe fit and a decreased eversion angle were also correlated with comfort rather than cushioning (Miller et al., 2000).

Movement Variability

It has been postulated that the effect of shoe design on impact forces is participant specific, and can not be generalized (Kersting and Bruggemann, 2006a). In some studies, where the effect of different midsole hardness on kinetics and kinematics parameters were investigated, a large participant variability in the kinetic and kinematic responses were documented (Dufek et al., 1991; Bishop et al., 2006; Kersting and Bruggemann, 2006b). Similar results were found with shoe inserts of different hardness (Nigg et al., 1998). It was suggested that the large variability was related to differences in lower limb and foot morphology, functional behaviour or sensitivity to external signals.

Other than differences in biomechanical parameters between the participants, the energy cost also appears to vary when runners wear shoes of different hardness (Nigg et al., 2003). According to the results from one study, there were participants who had a lower energy cost and reduced muscle activity of the vastus medialis before heel strike when wearing a harder (elastic) shoe, while others showed a lower level of muscle activity when wearing the softer (viscous) shoe. A third group, which the researchers considered as neutral, had no consistent results. Nigg et al. (2003) concluded that there are participant specific alterations in muscle activity and energy cost between running shoes.

Participant specific responses should therefore be taken into account when investigating biomechanical parameters or movement economy according to different shoe hardness.

Conclusion

Shoe proprieties, such as midsole hardness, flare, heel height edge and format of the heel sole, can affect kinematics and in some cases kinetics and muscle activity during running. Furthermore, comfort and biomechanics when running with shoes of differing hardness seems to vary according to the participants. Therefore, these characteristics should be considered and isolated in biomechanical studies on running shoes.

2.4.4 Running shoes and injury prevention

As indicated earlier, the role of running shoes in injury prevention was established initially as a reduction of impact force and control of pronation. However, more recent data appears to indicate that running shoes can alter sensory feedback and, therefore affect muscle activity.

2.4.4.1 Running shoes and reduction of impact forces

The importance of the reduction of impact force as a preventive mechanism for injuries was first postulated in a study in which sheep walked on a hard and soft surface for a period of two and a half years (Radin et al., 1982). In this study, the hard surface had a significant adverse effect on the sheep's knee articular cartilage, and the architecture of the underlying bone. This study was one of the first studies to support the notion that hard surfaces (extended to hard shoes) may predispose to injury.

To understand the mechanism of running shoes on reduction of impact forces and injury prevention, studies with cushioning inserts and running shoe age will be analysed. In a prospective study the use of a viscoelastic pad was effective in reducing the risk of Achilles tendon injuries (MacLellan and Vyvyan, 1981). There are also epidemiological studies where use of inserts on the incidence of injuries was studied in military recruits (Schwellnus et al., 1990; Mundermann et al., 2001). In the first of these the incidence of overall injury was similar between the insert and the control group; however, the incidence of tibial stress fractures was significantly lower in the group that used the insert (Schwellnus et al., 1990). In a more recent study a lower

incidence of stress fracture in military recruits was also documented in those recruits that wore shoe inserts (Mundermann et al., 2001). There was also an association between the insert, which was considered more comfortable, and the reduction of injury frequency. Although these results appear to be positive, the overall injury incidence of injury does not appear to decrease with the use of inserts. In a Cochrane review, where 5 trials (903 participants and 3006 controls) were included, the role of inserts in running injury prevention was reported as still unknown (Yeung and Yeung, 2001b).

The age of a running shoe may also be associated with running injury risk (Wen et al., 1997; Taunton et al., 2003). The rationale for this is that the cushioning properties of a running shoe may deteriorate with increasing running mileage (Cook et al., 1985a; Cook et al., 1985b; Verdejo and Mills, 2004). In laboratory tests with different models of running shoes it was shown that the shock absorbing capacity of shoes is reduced by 30% after about 800 km of running (Cook et al., 1985a). Therefore, it can be assumed that the increase in running shoe mileage may increase the risk of injury. In one study it was reported that uninjured runners changed their shoes on average every 7 months while injured runners do so every 10.8 months (Wen et al., 1997). However, in the same study, there was no correlation between injuries and the number of shoes alternatively worn (Wen et al., 1997). In one prospective study, 844 recreational runners followed a similar training programme (Taunton et al., 2003), running shoe age appeared to be protective but was also a source of a risk of injury, depending on the gender that was investigated.

Therefore there clearly is a need to further develop prospective studies to analyse the role of running shoes on impact forces and associated development of running injuries, possibly by examining a single injury, rather than all running injuries as a group.

2.4.4.2 Running shoes and the control of pronation

To our knowledge, there are no epidemiological studies, which have investigated the effect of anti-pronation shoes on the incidence of injuries. However, it has been

proposed that orthotics might be beneficial for the treatment of running injuries (D'Ambrosia, 1985)

There are two retrospective studies have shown the possible benefits of orthotics as an intervention to treat running injury (Blake and Denton, 1985; Gross et al., 1991). However, in a more recent review it was established that further prospective research is necessary to first establish the link between foot function and injury and then to investigate the efficacy of orthotic therapy on injuries (Razeghi and Batt, 2000).

A reduction of pronation trough variations of running shoes heel flare, edge and format and therefore improvement in foot stability has been the focus of most running shoe intervention studies *(Section 2.4.3.2 Effect of shoe proprieties on running biomechanics).* However, in a recent study (Nigg et al., 2006b), patients with osteoarthritis were shown to have a reduction in pain after 12 weeks when they wore an unstable shoe with a rounded heel sole. This result suggests that an increase in foot stability might not be appropriate for treating all running injuries.

2.4.4.3 Summary: Running shoes and injury prevention

There is some evidence that the use of insoles or reduced shoe age may be beneficial for injury prevention. However, according to a systematic review of level I studies there are insufficient studies to support this type of intervention (Yeung and Yeung, 2001a) (Table 2.7). Furthermore, studies on the role of anti-pronation shoes in injury prevention do not exist, and the efficacy on orthotics on injury prevention also needs further investigation (Table 2.8).

Table 2.7. The association between reduced impact force through different mechanisms and injury prevention using evidence-based medicine (EBM) criteria

Risk factor	Study information	Level of Evidence (I-V)
Use of insoles	**Positive association:**	
	Prospective studies: (Schwellnus et al., 1990; Mundermann et al., 2001)	I
	Case –control: (MacLellan and Vyvyan, 1981)	III
	No association:	
	Systematic review of level I studies: (Yeung and Yeung, 2001b)	I
Reduce shoe age	**Positive association:**	
	Prospective study: (Wen et al., 1997; Taunton et al., 2003)	I
	Negative association:	
	Prospective study: (Taunton et al., 2003)	I

Table 2.8. The association between control of pronation and injury prevention using evidence-based medicine (EBM) criteria

Risk factor	Study information	Level of Evidence (I-IV)
Use of orthotics	**Positive association:**	
	Retrospective study: (Blake and Denton, 1985; Gross et al., 1991)	II
	Expert opinion: (D'Ambrosia, 1985)	V
	No association:	III
	Systematic review level I-III studies: (Razeghi and Batt, 2000)	

2.5 General summary of the literature review

There is well established research related to the description of a "normal" running biomechanics. However, the literature is not so coherent when there is variation of the "normal" biomechanics, as a result of different running shoes or because of injuries.

There is some postulated evidence that altered biomechanics may be related to the aetiology of running injuries. However, other factors such as training errors and previous injuries seem to have a more established relationship to the aetiology of running injuries. More importantly the studies on biomechanics of running injury tend to relate anatomical measurements to some specific biomechanical characteristics during running (e.g. the association of pes planus and overpronation). Few studies have investigated the biomechanics of injured runners during running. More specifically relate to this thesis, there is only one study where the biomechanics of runners with Achilles tendinopathy were investigated (McCrory et al., 1999). Studies on biomechanics of runners with Achilles tendinopathy are limited to the study of kinetics and kinematics. To our knowledge there are no studies that have investigated the muscle activity of runners with Achilles tendinopathy.

It has recently been suggested that sensory feedback is an important mechanism to adjust biomechanical parameters during running (Kurz and Stergiou, 2003). Sensory feedback response seems to be subject specific and it has been shown that runners with patellofemoral pain appear to have lower intra-participant variability in biomechanical parameters compared with uninjured runners. However, the possible role of intra-participant and inter-participant variability as a risk factor for Achilles tendinopathy in runners has not been studied.

Finally, the biomechanics of running with different running shoes has been studied in uninjured runners, but to our knowledge, there are no studies that have studied the effect of different running shoes on biomechanical parameters in runners with Achilles tendinopathy.

CHAPTER 3: RESEARCH METHODOLOGY

In this chapter general details about the methodology used in the studies of this thesis will be described. However, as specific methods were used in some studies that require further explanation, these will be described in each specific research study chapter.

3.1 Participants

Thirty four uninjured runners (19 males and 15 females) and twenty one runners with Achilles tendinopathy (16 males and 5 females) participated on this study. The participants were recreational runners between the ages of 25 and 60 years old. To decide about the sample size a power calculation was done using data from previous studies in our laboratory which used similar outcome measurements. The statistical power was set at 80% and statistical significance 5% for the calculation.

All runners had a rearfoot running style as the mean ankle angle at initial contact for both groups was on average -11.1 ± 7.7° (negative values represent dorsiflexion, positive values plantarflexion). The characteristics of the participants are depicted in Table 3.1. Differences between the two groups in age, height, mass, years of running and training weekly distance were compared using the t test for independent variables. A more detailed analysis of the participant characteristics presented in Table 3.1 will be explored in Chapter 4. Study 1: *Training, Flexibility and Lower Limb Alignment Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*.

Table 3.1. Participant characteristics (Values are depicted as mean ± SD)

	Uninjured (N=34)	**Injured (N=21)**	**p value**
Male : Female	19:15	16:5	0.206
Age (yr)	37.0 ± 9.2	41.8 ± 9.7	0.217
Height (cm)	172.0 ± 9.0	177.8 ± 7.4	0.017
Mass (kg)	67.8 ± 10.1	77.6 ± 12.6	0.003
Years of running (yrs)	8.93 ± 6.0	13.6 ± 9.5	0.027

Training weekly distance (km)	43.0 ± 13.0	33.0 ± 18.0	0.021

Differences between genders were compared using Chi-Square. Statistical significance was accepted as $p \leq 0.05$.

Achilles tendinopathy in the injured runners was diagnosed by a sports physician at the Sports Science Institute of South Africa. In order for participants to be included in the Achilles tendinopathy group, the participants were assessed and fulfill all of the following clinical diagnostic criteria for non-insertional Achilles tendinopathy (Kader et al., 2002): 1) gradual progressive pain over the posterior lower leg (Achilles tendon area), 2) early morning pain and stiffness, 3) a history of swelling over the Achilles tendon area, 4) tenderness to palpation over the Achilles tendon, 5) palpable nodular thickening over the affected Achilles tendon, and 6) a positive "shift" test (movement of the painful nodular area with plantar-dosiflexion). An example of the Clinical Diagnosis sheet is presented depicted in Appendix 2. Furthermore, in 48% of the injured runners, the diagnosis was also confirmed by a soft tissue diagnostic ultrasound scan, using criteria including abnormal tendon structure, nodular thickening, altered fibre orientation and hypoechoic areas (Alfredson, 2003; varez-Nemegyei and Canoso, 2006).

To be included in the uninjured group, the runners had to be uninjured for at least 2 years. In both groups, the runners had to run for at least 3 years and have no current or past history of neurological disorders, diabetes mellitus or physical deformities. Physical deformities was defined a severe distortion of body shape as a results of genetic defects, severe injury or past surgery (loss of limb or severe anatomical abnormality). The criteria were largely subjective and were determined by history and clinical assessment (in particular by assessment of the lower and upper limbs). The injured runners all had grade I (pain only after running) or grade II pain (pain during running but not restricting running) (Noakes, 2001b). The reason for choosing participants with these grades of pain was that runners were tested in a biomechanics laboratory where they were required to run pain free as part of the experimentation.

A participant information sheet was provided for each participant explaining the nature of the study and the procedures involved (Appendix 3). Written informed consent was obtained from all participants before the study (Appendix 4). The study

was approved by the Research Ethics Committee of the Faculty of Health Sciences at the University of Cape Town in South Africa (Appendix 5).

All the runners were requested to complete a self-administered questionnaire (Appendix 6). The questionnaire was divided in four sections as follows: personal details, medical history, injury history and training history. The responses to these questionnaire sections are further explored in Chapter 4. Study 1: *Training, Flexibility and Lower Limb Alignment Variables as Risk Factors Associated with Achilles Tendinopathy in Runners.*

3.2 General experimental procedure

Each participant was required to report to the laboratory on one occasion for testing. All the testing took place at the Gait Analysis Laboratory located in the Sports Science Institute of South Africa (SSISA), Newlands, Cape Town, South Africa.

The height and body mass of participants' were measured with the laboratory scale (Seca, Model 708, Germany). An anthropometrical beam calliper (Harpenden Anthropometer, Corswell, UK) and a tape (Mabbis, Illinois, USA) were used to measure the anthropometric variables. The measurements included pelvic width and bilateral measurements of the thigh, calf and foot segments. A more detail justification of the measurements is provided in Appendix 7 (Vaughan et al., 1999). These anthropometric data were used later in regression equations of the model parameters during the process of data analysis to predict masses and moment of inertia of lower extremity segments.

Fifteen retro-reflective markers from the modified Helen Hayes marker set (Vaughan et al., 1999) were used to collect kinematic data. The markers were attached on the skin with double sided tape in different anatomical positions (described in Appendix 8). The markers reflected light, which originated from multiple light emitting diodes circumferentially arranged about the lens of each of the six digital cameras Oxford Metrics Vicon System 370 Version 2.5 (Oxford Metrics Ltd, Oxford, United Kingdom). The cameras collected the data at a frequency of 120Hz and conveyed data

by underfloor cabling to a data station, which converted it to a digital form. Before data collection, the six cameras were calibrated with the calibration L frame and a wand for the static and dynamic calibration respectively.

Ground reaction force data were collected with an Advanced Mechanical Technology, Inc. (AMTI® Newton, MA, USA) force plate (1000Hz), which was embedded within a six meters long pathway. A carpet over the force plate disguised the exact location of the force plate in an attempt to prevent subjects from altering their gait. Analogue data were conveyed from the force plate to a data station and personal computer.

Surface electromyography (EMG) signals were recorded through Telemyo Noraxon EMG Systems (Noraxon USA, Inc) at a sampling rate of 2000 Hz. EMG signals were detected from each muscle by two surface triode electrodes (Thought Technology Triode MIEP01-00, Montreal, Canada). Surface electrodes were placed over the visual midpoint of the contracted belly of the muscle and aligned along a line approximately parallel to the direction of the muscle fibre (Kamen and Caldwell, 1996). Before electrode placement, the skin was shaved and cleaned with an alcohol wipe. The following muscles were tested: tibialis anterior (TA), lateral gastrocnemius (LG), peroneus longus (PE), rectus femoris (RF), biceps femoris (BF) and gluteus medius (GM) muscles. A single electrode (ground electrode) was placed over the patella . The cables and amplifiers were taped to the skin to minimise movement artefacts. The electrodes were placed on the right limb for the uninjured runners and on the affected limb for the injured runners. The location of the reflective markers and EMG electrodes on a participant are shown in Figure 3.1.

Figure 3.1 Reflective markers and EMG electrodes position

The EMG, kinetic and kinematic data were collected simultaneously during the recording process through Oxford Metrics Vicon System and therefore synchronized. The synchronization occurred through Workstation ® (Oxford Metrics, England). The EMG receiver (Noraxon Inc, USA) was connected to Workstation which auto adjusted the setting of the EMG to 1920 Hz to agree with the Vicon cameras frequency (120 Hz) and also with the force place (1920Hz).

By default all trials were conducted with the participant wearing a standard neutral running shoe (Rainha Athens, Alpargatas Inc; shore A 40), sizes 6 to 12 and no socks or orthotics. The runners first underwent a familiarization trial during which they performed an easy jog warm-up around the lab for 5 minutes at a self paced speed. After the familiarization trial, the runners were instructed to run 10 trials at a self selected speed on the 10 m pathway of the gait laboratory. To promote a more natural running style during the short 10m run, the participants were asked to continue running during the 10 trials. A trial was considered valid when the runner's entire foot (injured runners – injured foot, uninjured runners – right foot) made contact with the force plate and there was no alteration in the running style as judged subjectively by the investigator.

3.3 Data Analysis

Five valid trials were selected for further analysis. The trials were selected based on the quality of the data (eg. no missing markers and a good EMG signal). A list of EMG data that were excluded from the different chapters is presented in Appendix 9. The data were processed for one stride length (one step before and one step after the force plate).

3.3.1. Data Analysis of Kinetic and Kinematic variables

The data collected from Oxford Metrics Vicon System were initially labelled in the Workstation® program (Oxford Metrics, Oxford, England) using the Gait Lab marker set parameters (.mkr). After the labelling process, the C3D files were exported to Body Builder® program (Oxford Metrics, Oxford, England), where the data of a complete stride were cut 5 frames before and 5 frames after the toe off, by visual interpretation of heel strike and toe-off. The data were then processed in Body Builder® using the model parameter, which contained the anthropometrical data of the participant (.mp), the marker parameter (.mkr) and the Gait model (.mod) (Tabakin, 2000). Kinematic data was filtered with the weighted average filter, and a low pass digital filter was applied for the kinetic data. After this process, the data were exported as text files for analysis in Excel® (Microsoft Corporation, Redmond, USA). The data were then reduced more precisely for a stride length using the horizontal displacement of the heel marker (Figure 3.2). First heel strike was defined as the point where the heel marker presented a constant value defined in the Figure 3.2 as HS1 (heel strike one) and the next heel strike was defined as the point at the end of the curve where heel marker presented a stable value (HS2 = heel strike 2).

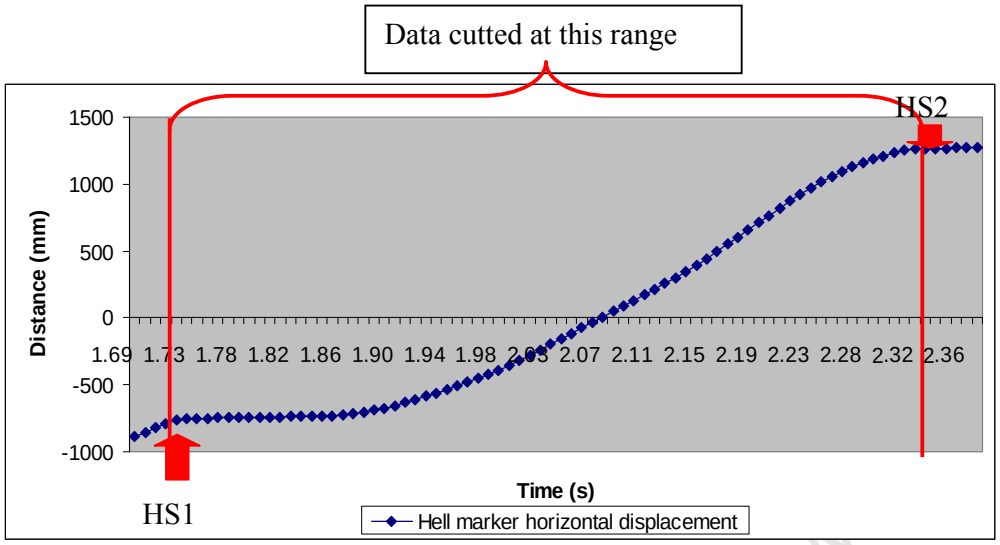

Figure 3.2. Typical displacement-time graph of the heel marker on a single running trial.

Abbreviations: *HS1 = Heel strike one; HS2 = Heel strike two*

The Excel files were temporally normalized to 51 data points (0 to 100% gait cycle, intervals of 2%) by a set program in Matlab™ (Math Works, Natick, MA). The same program also averaged and calculated the standard deviation for the five selected trials. Selected kinetics and kinematics were then analysed in Excel. The decision to use the average of five trials for further analysis instead of analysis of an individual trial was because the five trials is a better representation of the individual running style.

3.3.1.1 Temporal Distance Variables

The running velocity (m/s) was determined by the sacrum retroreflective marker attached to each runner. The horizontal velocity was calculated through the movement of the marker on the x-axis of the global reference system of the laboratory for each trial and the average of five trials was estimated.

The horizontal displacement of the heel marker was chosen to determine the stride length (m), while the time to complete a stride was used to calculate the stride frequency (strides/s). Similarly, contact time was described by the intial contact time

(s) of the heel marker on the floor to the final contact of the toe marker on the floor on the x-axis.

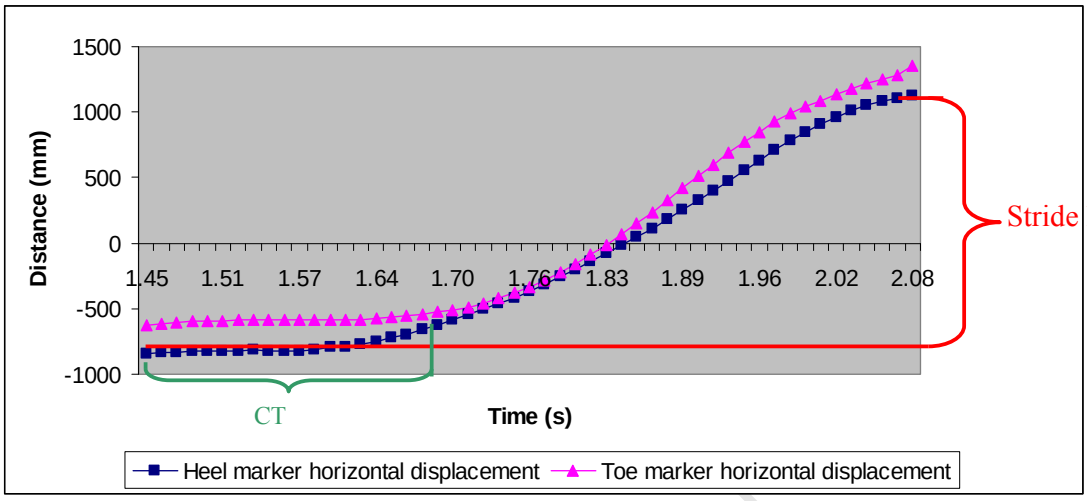

Figure 3.3. Typical displacement-time graph of the heel and toe markers averaged for five trials.

Abbreviations: *CT = contact time*.

The vertical body oscillation (m) was determined by the first vertical displacement of the sacral marker in the z-axis (Figure 3.4).

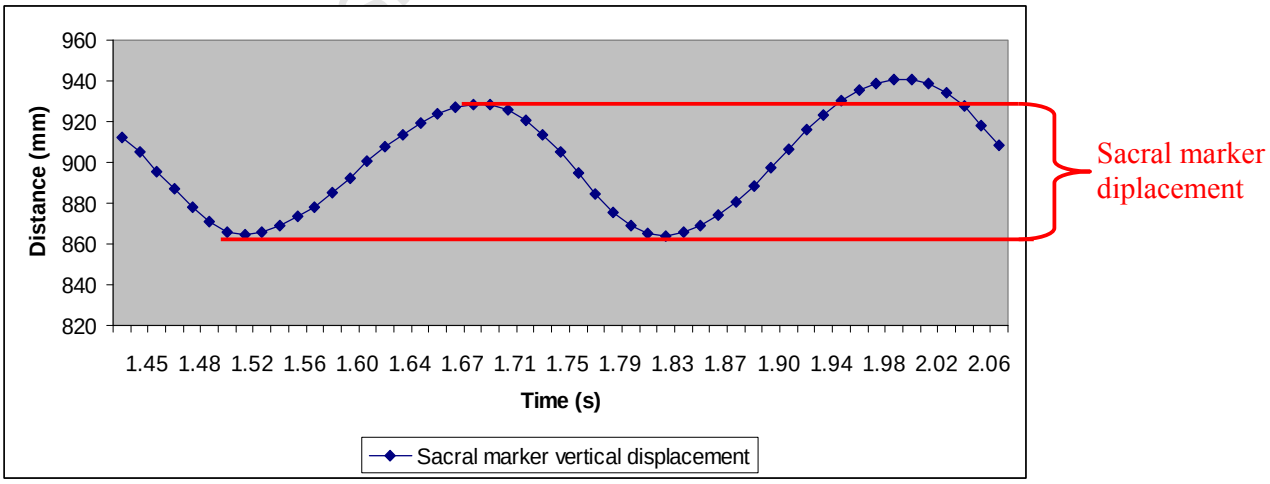

Figure 3.4. Typical displacement-time graph of the sacral markers averaged for five trials.

3.3.1.2 Kinetic variables

The vertical ground reaction force (GRF) variables that were selected for analysis included: 1. vertical impact force peak (VIF) defined by the first peak just after heel strike, 2. vertical propulsive force (VPF) defined by the second peak after heel strike and 3. vertical loading rate (VLR) defined as the VIF divided by the time to reach the VIF (Hargrave et al., 2003) (Figure 2.3, *A Review of literature*).

The anterior-posterior GRF variables that were selected included: 1. Horizontal braking force (HBF) defined by the negative peak and 2. Horizontal propulsive force (HPF) defined by the positive peak (Figure 2.2, *A Review of literature*). All kinetic variables were expressed in multiples of body mass (BW).

3.3.1.3 Kinematic variables

Sagittal and frontal plane kinematics were processed for the hip, knee and ankle joints. A more detail description of each variable is presented in Table 3.2 and a suitable graphic depiction is illustrated in Figures 3.5 and 3.6.

Table 3.2 Temporal distance, kinetics and kinematics variables that were included in the analyses

Symbol	Variable	Definition
Hic	Hip angle at heel strike	Hip flexion angle at heel strike
Hto	Hip angle at toe off	Hip extension angle at toe-off
HROM	Hip range of motion	Range of motion of hip flexion from heel strike to toe-off
THR	Time of maximum hip rotation	Time of maximum hip internal rotation at stance phase
KswE	Knee angle in terminal swing phase	Maximum knee extension angle at terminal swing phase
Kic	Knee angle at initial supporting surface contact	Knee flexion angle at heel strike
Kst	Knee angle in midstance	Maximum knee flexion angle in stance phase
KROM	Range of motion of knee flexion	Range of motion of knee flexion from heel strike to midstance
Asw	Ankle angle in terminal swing phase	Ankle angle at the point of maximum knee extension at terminal swing phase
Aic	Ankle angle at heel strike	Ankle dorsiflexion angle at heel strike
Ast	Ankle angle in midstance	Maximum ankle dorsiflexion angle in stance phase
Δβev	Delta eversion angle at contact phase	Difference between initial foot inversion and maximum foot eversion
Time Bev	Time to peak eversion	Time of peak eversion on stance phase

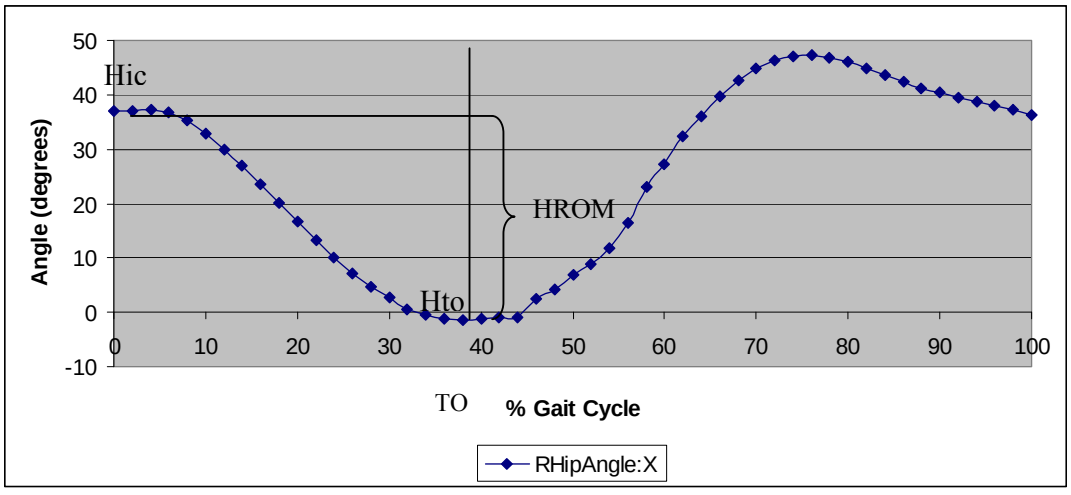

Figure 3.5. Typical hip kinematics during running - sagittal plane.
Abbreviations: *TO = toe –off, Hic = hip angle at initial contact, Hto = hip angle at toe-off, HROM = hip range of motion.*

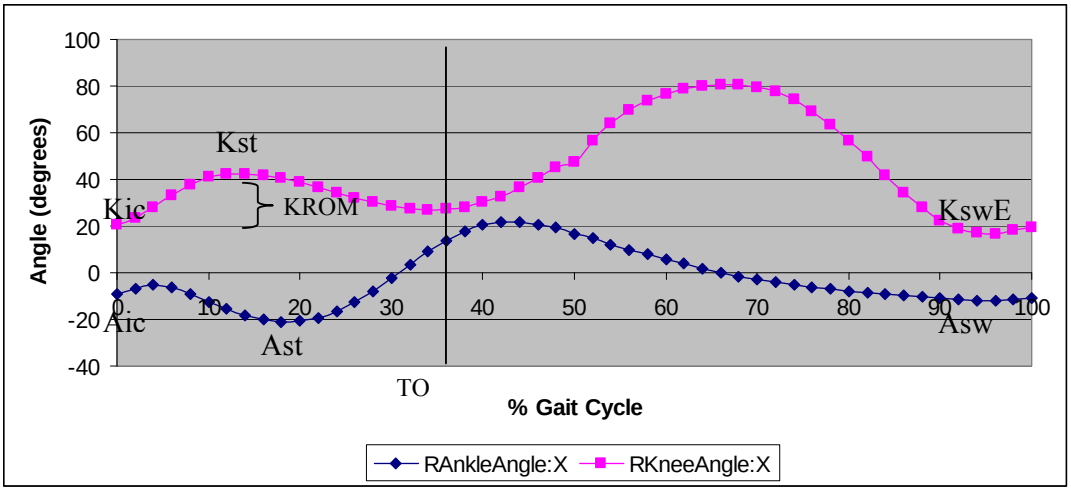

Figure 3.6. Typical knee and ankle kinematics during running - sagittal plane. Abbreviations: *TO = toe-off, Kic = knee angle at initial contact, Kst = knee angle at stance phase, KROM = knee range of motion, Aic = ankle angle at initial contact, Ast = angle at stance phase.*

3.3.2. Data Analysis: EMG parameters

The EMG data were collected through the Vicon System and the analog data (.vad) were exported to Myo Research Software (Noraxon Inc, USA) for further analysis. The EMG data were pre-amplified by a band-pass filter with a cut-off frequency at 15 and 500 Hz. Root mean square amplitude (RMS) of all EMG signals was calculated over consecutive periods of 50 ms. The mean dynamic method was applied to normalize the EMG (Shiavi et al., 1987). The decision to use mean dynamic method for normalization, instead of other methods suggested by the literature (Burden et al., 2003) was determined through a pilot study which was conducted in the laboratory. The results of this study are presented in Appendix 10. Values were expressed as a percentage of the mean RMS EMG.

Integrated EMG (IEMG), which represents the total accumulated EMG activity over a period of time (Kamen, 2004), was calculated as the area under the curve for the 100 ms before heel strike (Pre IEMG) and 100 ms after heel strike (Post IEMG). The data were processed for each individual trial and the results of five trials per participant were averaged. The decision to analyse the Pre and Post IEMG activity using individual trials instead of the temporally normalized averaged of five trials, as the followed procedures for all the others kinetics, kinematics and EMG variables, was

because this data are specifically associated with a certain time period (100ms) and the time normalization would affect the results.

The five selected EMG data per participant which were processed using Myo Research software were then exported as an Excel file (Excel® Microsoft Corporation, Redmond, USA), where data were reduced to only one stride length. The identification of the stride length for each singular trail was based on the time of a stride lenght determined by the processed kinematics data previously described before time normalization (section 3.3.1. *Data Analysis of Kinetic and Kinematic variables,* Figure 3.2). This was only possible because kinetic, kinematic and EMG data were synchronized during data collection. The selected EMG data of a stride length were then temporally normalized in 51 data points (0 to 100% gait cycle, intervals of 2%) and the average and standard deviation of five trials were processed by a set program in Matlab™ (Math Works, Natick, MA).

The averaged data were then copied to GraphPad Prism v5 Software Inc. (Graph Pad, San Diego, CA) where the area under the curve (IEMG) was processed for stance phase and the whole running cycle. The identification of stance phase was based on the temporal distance parameters data after time normalization previously processed (Section 3.3.1.1 *Temporal Distance Variables*, Figure 3.3). Furthermore, peak EMG activity and percentage of gait cycle where the peak EMG activity occurred were also calculated for the same periods (stance phase and whole gait cycle) on GraphPad Prism v5 Software Inc.

3.4 Repeatability of the kinetic, kinematic and EMG data

To assess the repeatability of the data, coefficient of variation (CV) and variance ratio (VR) were calculated over the entire running cycle for kinetics, kinematics and EMG data for each individual over 5 strides (1 stride per trial). The CV is normally applied to assess repeatability of kinetic and kinematic data (Menz et al., 2004; Queen et al., 2006) while the VR is commonly used to assess repeatability of EMG data (Kadaba et al., 1985; Burden et al., 2003; Bogey et al., 2003). The following equation was applied for the CV:

$$CV = \frac{\sqrt{\frac{1}{k}\sum_{i=1}^{k}\delta_i^{\,2}}}{\frac{1}{k}\sum_{i=1}^{k}\left|\overline{Xi}\right|}$$

where, k is the number of time intervals over the gait cycle (i.e. 51 data points), X_i is the mean of the kinetics, kinematics and EMG values at the i^{th} interval calculated over five trials for the 55 participants (21 injured and 34 uninjured), σ_i is the standard deviation of X_i for the EMG, kinetics and kinematics values.

The variance ratio (VR) equation used was:

$$\mathrm{VR} = \frac{\sum_{i=1}^{k}\sum_{j=1}^{n}(X_{ij}-\bar{X}_i)^2/k(n-1)}{\sum_{i=1}^{k}\sum_{j=1}^{n}(X_{ij}-\bar{X})^2/(kn-1)}$$

where, k is the number of time intervals over the gait cycle (i.e. 51 data points), n is the number of trials in this case five, X_{ij} is the EMG, kinetic or kinematics value at the i^{th} interval for the j^{th} trial. X_i is the mean of EMG, kinetic or kinematic values at the i^{th} interval over the j gait cycle and $\overline{X}$ is the mean of EMG, kinetic or kinematic values, i.e.: $\overline{X} = 1/k \sum_{i=1}^{k} \overline{X}i$. These equations were described by Burden et al., 2003.

The results of CV and VR are presented in Table 3.3.

Table 3.3. The coefficient of variation for kinetic, kinematic and EMG variables over a running cycle (N= 59) (Values are depicted as mean ± SD).

Parameters	CV	VR
Anterior-posterior GRF	0.60 ± 0.18	0.18 ± 0.17
Vertical GRF	0.26 ± 0.09	0.06 ± 0.07
Hip Flexion-Extension Angle	0.12 ± 0.13	0.05 ± 0.11
Knee Flexion-Extension Angle	0.14 ± 0.26	0.06 ± 0.13
Ankle Plantar Flexion- Dorsiflexion Angle	0.30 ± 0.26	0.10 ± 0.12
Ankle Inversion – Eversion Angle	0.33 ± 0.24	0.19 ± 0.15
TA EMG	0.34 ± 0.18	0.38 ± 0.20
PE EMG	0.45 ± 0.18	0.30 ± 0.23
LG EMG	0.48 ± 0.22	0.30 ± 0.24
RF EMG	0.46 ± 0.17	0.31 ± 0.24
BF EMG	0.41 ± 0.16	0.39 ± 0.21
GM EMG	0.43 ± 0.15	0.44 ± 0.25

Abbreviations: *GRF = ground reaction force, EMG = electromyography, TA = tibialis anterior, PE = peroneus longus, LG = lateral gastrocnemius, RF = rectus femoris, BF = biceps femoris, GM = gluteus medius.*

The coefficient of variation for the kinetic and kinematic data are presented in Table 3.4, were in some cases higher than a previous study (Queen et al., 2006). However, in that study the CV was calculated for discrete variables (e.g. peak angles and peak forces), while in this study it was calculated throughout the whole gait cycle curve. Furthermore, this study investigated a larger and a more heterogeneous sample compared to the previous study, which studied a population of only 12 uninjured runners (Queen et al., 2006). Nevertheless, the main differences between the studies were in the kinetic variables while the joint kinematics were more similar.

Variance ratio (VR) has been suggested as a method to measure repeatability of waveforms (Kadaba et al., 1985). Low values of VR indicate high repeatability and values of VR below 0.30 are considered to present good repeatability (Kadaba et al., 1985; Bogey et al., 2003). Although most of the studies have investigated the repeatability of discrete variables (Diss, 2001; Ferber et al., 2002), other authors (Queen et al., 2006) suggest that the waveform pattern of the kinetic and kinematic

variables of a running cycle could provide more information about the reproducibility of the continuous measures. In this present study, all of the kinetic and kinematic data had a VR below 0.19, which indicate good repeatability.

The VR of EMG data were in the range of 0.30 to 0.44. These values were lower than reported in some studies (Pierotti et al., 1991; Burden et al., 2003) but higher than others (Kadaba et al., 1985; Bogey et al., 2003). All these studies have investigated EMG variability during walking gait, which has to be differentiated from running. During running there are higher movement artefacts due to cable and amplifier movements. Furthermore, comparison of the data with the literature can be difficult as variability seems to alter according to the method of EMG normalization (Burden et al., 2003), type of shoe (Kurz and Stergiou, 2003) and injury condition (Hamill et al., 1999).

A more in-depth investigation of intra and inter-participant variability will be presented in Chapter 6 Study 3: *Variability in Biomechanical (kinetic, kinematic and muscle activity) Parameters as Risk Factors Associated with Achilles Tendinopathy in Runners.*

3.5 Assumptions and limitations of the general methodology

It is important to point out that in the development of the methodological process some assumptions and limitations need to be taken into consideration.

Firstly, the decision to place the electrodes only on the right limb of the uninjured group was based on results from other studies, in which no difference between left and right limbs for kinetics and kinematics parameters was found in uninjured runners (Cavanagh, 1987). However, other studies do show some asymmetry between dominant and non-dominant legs for EMG parameters during walking (Ounpuu and Winter, 1989). In the present study, eight of the twenty one injured participants had their injury on the left leg. A limitation of this study was therefore that left and right side measurements were not balanced in the control group to match the injured population.

Secondly, day-to-day variability was not tested in this study. However in a previous study on repeatability of kinetic, kinematic and EMG data during walking in normal adults it was observed that clinical decisions can be based on the results of a single gait evaluation (Kadaba et al., 1989). Therefore, conclusions can be drawn for a one day trial.

Thirdly, it has been stated that knee kinematics may vary between male and female participants (Malinzak et al., 2001), although similar sagittal plane kinematics between gender were found in another study (Ferber et al., 2003). In this study, there were no statistical differences between the male and female distribution (Table 3.1), therefore it can be assumed that the sample sizes was homogeneous in relation to gender. However, to explore this decision better, further statistical tests were performed for the same variables in a smaller and more homogeneous population (18 male: 7 female in each group). These results are presented in Appendix 11 and show some differences compared with the larger sample size (e.g.: peroneus longus 100 ms after heel strike and stance phase significantly higher on the injured group). Although some results may be different with a smaller and more homogeneous sample, it was decided to maintain the statistical power of a larger population since there were no statistical differences between genders.

CHAPTER 4. STUDY 1: TRAINING, FLEXIBILITY AND LOWER LIMB ALIGNMENT VARIABLES AS RISK FACTORS ASSOCIATED WITH ACHILLES TENDINOPATHY IN RUNNERS

4.1 Introduction

Several extrinsic factors and intrinsic risk factors have been listed as possible causes of running injuries (van Mechelen, 1992; Rolf, 1995; Krivickas, 1997; Neely, 1998), and these were classified and reviewed, using evidence base medicine (EBM) criteria (Obremskey et al., 2005) in Chapter 2 (Section 2.2 *A Review of the Epidemiology of Running Injuries*) of this thesis. The data presented in Chapter 2 showed that extrinsic risk factors for running injuries are training errors, running surface, stretching, warm-up and cross-training. However, only for training errors, more specifically increased training distance, is there strong evidence to supporting the association between the risk factor and the development of a running injury (Lysholm and Wiklander, 1987; Walter et al., 1989; Macera et al., 1989; Yeung and Yeung, 2001a; van Gent et al., 2007).

Postulated intrinsic risk factors for running injury are a history of a previous injury, running experience, age, gender, body weight, biomechanics, lower limb abnormalities, flexibility and muscle strength (Obremskey et al., 2005). In Chapter 2, intrinsic risk factors for running injuries were also reviewed, using evidence based medicine criteria. The only intrinsic risk factor that was strongly associated with running injury risk was a history of a previous injury (Walter et al., 1989; Macera et al., 1989; Taunton et al., 2003), supporting the results of recent published systematic review (van Gent et al., 2007).

The focus of this thesis is on Achilles tendinopathy, therefore the intrinsic and extrinsic risk factors associated with Achilles tendinopathy injury were also reviewed in Chapter 2, section 2.3 *A Review of the Epidemiology of Achilles Tendinopathy*. The main findings of this review showed that Achilles tendinopathy seems to be more common in male runners over 35 years old, and may be associated with muscle

weakness or muscle imbalance as well as poor flexibility of the gastrocsoleus muscle group. Furthermore, training errors and limited warm–up were also identified as possible extrinsic risk factors associated with Achilles tendinopathy (Smart et al., 1980; Clement et al., 1984; Alfredson et al., 1998b; Dingwell et al., 1999; McCrory et al., 1999; Alfredson and Lorentzon, 2000; Milgrom et al., 2003; Barr and Harrast, 2005).

The aim of the first research study in this thesis was to explore the relationship between some of these previously reported intrinsic and extrinsic risk factors for running injuries in a group of runners with Achilles tendinopathy. Specifically, the aim was to compare training parameters, injury history, body composition, flexibility and lower limb alignment variables between uninjured runners and runners with Achilles tendinopathy. It was hypothesised that runners with Achilles Tendinopathy will have a history of increased training volume, a history of previous injury, and specific anatomical lower limb alignment characteristics that will distinguish them from uninjured runners.

4.2 Methods

4.2.1 Participants

The details of the recruitment and specific participant characteristics for this study have already been described in the general methodology Chapter 3- *Research Methodology*.

4.2.2 Procedures

The following data were collected from all the participants in the injured and uninjured groups.

4.2.2.1 Questionnaire data

As described previously mentioned (Chapter 3 – *Research Methodology*) all the runners completed a self-administered questionnaire (Appendix 6). The questionnaire provided data on personal details, medical history, injury history and training history. The following selected variables were further analysed for the injured participants:

1.Severity of injury (measured by Grade of Injury); 2. Self reported possible causes of injury; 3. A history of a previous injury; and 4. Type of previous injury.

The following additional self-reported training variables were also analysed and compared between the injured and uninjured participants: 1. Years of running; 2. Competitive level; 3. Running Surface; 4. Number of running shoes and kilometres run per shoes; 5. Use of orthotics; 6. Cross- training; 7. Stretching habits; 8. Warm-up habits and 9. Average weekly running distance.

The decision to select these variables was based on the findings from the data presented in the literature review (Chapter 2) on possible risk factors for running injuries, in particular Achilles tendinopathy (Walter et al., 1989; Macera et al., 1989; van Mechelen, 1992; Macera, 1992; Taunton et al., 2003). The average weekly running distance data were analysed only over the three months preceding the visit in order to limit recall bias (it was assumed that most of runners could not necessarily remember their training distances for periods longer than 12 weeks). Training intensity was not addressed because most of the participants were not able to accurately recall their training intensity. Furthermore, participants who provided the information had different methods to record training intensity, some use more objective methods such as heart rate, while others use a more subjective method such as perception of effort (e.g. light, moderate and hard). Therefore, it was difficult to have a uniform measurement of training intensity which could be analyzed for all the participants.

4.2.2.2 Body Composition and Flexibility

4.2.2.2.1 Body Fat percentage

Skinfolds thicknesses were measured on the right side of the body with the participant in a standing position. A Lange skinfold caliper (Beta Technology, Santa Cruz, California, USA) was used to measure the skinfold (SF) thickness of the triceps, biceps, subscapular and suprailiac as described below (American College of Sports Medicine, 1993):

a. Triceps: vertical fold in the mid point between the acromiom and olecranon process.

b. Biceps: vertical fold over the belly of the biceps femoris, 1 cm above the level of the triceps skinfold.

c. Subscapular: diagonal fold, 2 cm along the inferior line of the scapula

d. Suprailiac: diagonal fold, on the anterior line of the axillary line, superior to the iliac crest line.

The body density was estimated from the following equations for male and female participants (Durnin and Womersley, 1974):

Men - Body Density:
Age < 20: 1.1620 - 0.0630 * Log10 (sum of 4 skinfolds)
Age < 30: 1.1631 - 0.0632 * Log10 (sum of 4 skinfolds)
Age < 40: 1.1422 - 0.0544 * Log10 (sum of 4 skinfolds)
Age < 50: 1.1620 - 0.0700 * Log10 (sum of 4 skinfolds)
Age >= 50: 1.1715 - 0.0779 * Log10 (sum of 4 skinfolds)

Women - Body Density:
Age < 20: 1.1549 - 0.0678 * Log10 (sum of 4 skinfolds)
Age < 30: 1.1599 - 0.0717 * Log10 (sum of 4 skinfolds)
Age < 40: 1.1423 - 0.0632 * Log10 (sum of 4 skinfolds)
Age < 50: 1.1333 - 0.0612 * Log10 (sum of 4 skinfolds)
Age >= 50: 1.1339 - 0.0645 * Log10 (sum of 4 skinfolds)

After the calculation of body density, body fat were calculated from the following equation (Siri, 1956):

Body fat [%] = 495 / body density – 450

4.2.2.2.2 Sit and reach test

The sit and reach test assesses flexibility of the posterior leg and trunk muscles and has been validated as a suitable test (Hui and Yuen, 2000). The sit-and-reach test was

performed using well-defined standard procedures (American College of Sports Medicine, 2000), The participant performed a guided stretching of the posterior muscle groups using these two exercises: 1) Exercise 1: in a standing position, the participant crossed the right foot in front of the left and lowered the forehead in front of the right knee by bending the hip. This position was held for 10 seconds and than repeated by crossing the left foot in front of the right foot; 2) Exercise 2: the participant sat on the floor, one leg straight and the other bent at the knee and positioned the sole of the foot against the opposite leg. The participant extended the arms and reached forward as far as possible over the straight leg and held this position for 10 seconds and than repeated it once again.

The participant removed his/her shoes and sat on the floor with the knees extended and the feet against the edge of the box and maintained 15 cm apart. With the arms stretched and the hands in a parallel position, the participant reached as far as possible with both hands and without flexing the knees. The score (in cm) was recorded as the most distant point reached by the fingertips. The best of three trials was recorded (American College of Sports Medicine, 2000).

4.2.2.3 Lower limb alignment

Each participant underwent a series of standard anthropometrical measurements to assess lower limb alignment. The examiner (Liane Azevedo) was first trained by an experienced podiatrist to perform all the measurements. Each measurement was performed twice and an average of two measurements was used in the data analysis. The description of these measurements is as follows:

4.2.2.3.1 Rearfoot alignment

This test measures the alignment of the rearfoot in relation to the calf in a weight bearing position. The rearfoot alignment angle was measured when one of the arms of the goniometer was placed on the line of calf to the Achilles tendon and the other arm on a line bisecting the calcaneus (Houglum, 2005). It has been reported that this measurement of rearfoot angle has an intra-examiner reliability of 0.88 and inter-examiner reliability of 0.86 (Jonson and Gross, 1997).

4.2.2.3.2 Pronation (heel eversion) angle 45°

This test measures the pronation (or heel eversion) angle when the knee flexes at 45° (Kannus, 1992) In a weight bearing position starting in full knee extension and the feet facing forward, the participant was asked to perform a squat to 45° flexion. The pronation (or heel eversion) angle was measured with a goniometer that was positioned on the visually estimated midline of the calcaneus and the arm of the goniometer is aligned to the tendon (Viitasalo and Kvist, 1983). The intra-examiner reliability for this test has been reported as 0.72-0.95 (Levinger et al., 2006).

4.2.2.3.3 Quadriceps (Q) angle

The quadriceps (Q) angle is the frontal plane angle of the resultant force of the quadriceps on the patella and tibial tuberosity (Heiderscheit et al., 1999). The Q angle was measured in both limbs. The goniometer axis was placed at the centre of the patella and one of the goniometer arms was aligned to the anterior superior iliac spine while the other arm was aligned to the tibial tuberosity. The angle between the intersection of the anterior superior iliac spine and the tibial tuberosity was measured and recorded as the Q angle (Heiderscheit et al., 2000). Q angle reliability measurements have been reported as 0.17-0.29 for inter-examiner reliability and 0.14-0.37 to intra-examiner reliability (Greene et al., 2001).

4.2.2.3.4 Standing foot angle

The standing foot angle (SFA) is the angle between the medial malleolus, navicular prominence and the first metatarsal head This measurement is taken with the participant standing in a weight bearing position, using a goniometer (Sommer and Vallentyne, 1995). In a study with injured population inter-examiner reliability of this tests showed a significant difference of 2.5° between examiners (Sommer and Vallentyne, 1995). However, the authors pointed out that this difference is equivalent to the smallest increment that can be measured using a conventional goniometer.

4.2.2.3.5 Navicular drop

The navicular drop measures the difference between the vertical distance of the navicular in non-weight-bearing and weight bearing position (Loudon et al., 1996).

The navicular drop is measured with the participant initially in a seated position (non-weight bearing). The vertical distance between the most prominent aspect of the medial navicular bone and the floor is then measured. The participant was then asked to stand (weight-bearing position) and the same measurement was performed. For this measurement, interexaminer reliability was reported as excellent (0.93) (Piva et al., 2006)

4.2.2.3.6 Forefoot alignment

Forefoot alignment measures the position of the first and the fifth metatarsal bone (Kannus, 1992).Forefoot alignment was measured with the participant lying in a prone position and the foot extended about 20 cm over the edge of the table to allow free movement of the foot and ankle during measurements. The leg that is not being measured was placed in knee flexion and external hip rotation. The amount of forefoot varus was measured by the amount of lift between the first and fifth metatarsal head in millimeters when the foot was placed in a subtalar joint neutral position using standard clinical techniques (Kannus, 1992). Inter-examiner reliability for this test has been reported as 0.68 (Astrom and Arvidson, 1995).

4.2.2.3.7 Subtalar joint passive range of motion for eversion and inversion

The passive range of motion (PROM) of eversion and inversion of the subtalar joint is determined by the angle between the lower leg and the calcaneus when the heel is placed in maximum eversion and inversion respectively (Elveru et al., 1988).

The participant was placed in a prone position with the foot extended about 20 cm over the edge of the table while the other leg was with the knee in a flexed position and the hip externally rotated (as in section 4.4.3.6). A line, midway between the medial and lateral borders on the posterior lower leg was drawn along the back of the lower leg. The examiner than located the medial and lateral prominence of the talus. The determination of the subtalar joint neutral (STJN), was at the position where the examiner was able to palpate the prominences of the talus medially and laterally equally. The STJN was measured by the angle of the two arms of a goniometer that were aligned to 1) the longitudinal midline of the calcaneus and 2) the line drawn on

the lower leg (Elveru et al., 1988). The inter-examiner reliability of this test has been reported as 0.58 for inversion and 0.36 for eversion (Astrom and Arvidson, 1995).

4.2.2.3.8 Knee range of motion

Knee range of motion was measured as the angle of the knee from full extension to maximal knee flexion. Knee range of motion was measured with the participant lying in a supine position. The fulcrum of the goniometer was centered on the lateral femoral condyle. One arm of the goniometer was aligned to a line form the lateral femoral condyle to the greater trochanter of the femur while the second arm of the goniometer was aligned along the fibula from the lateral femoral condyle to the lateral malleolus (Springhouse Corporation Staff, 2001). With the assistance of the examiner, the measurement started with the leg fully extended and the leg was then moved to maximal knee flexion. The intra-examiner reliability for this measurement has been reported as 0.99 and inter-examiner reliability as 0.90 (Watkins et al., 1991).

4.2.2.3.9 Subjective assessment and classification of foot type

The foot type of each participant was assessed subjectively during weightbearing, and the configuration of the longitudinal arch was classified as pes planus, cavus or neutral (Kannus, 1992; Lun et al., 2004). Inter-examiner reliability for this visual assessment of foot type has been reported as 0.72 (Dahle et al., 1991).

4.2.2.3.10 Leg length discrepancy

Leg length discrepancy is the difference in length between the lower extremities. It is well established that there are a number of techniques to measure leg length discrepancy. In this study, leg lengths were measured with the participant lying in a supine position after the pelvis alignment was placed on a neutral position (by fully flexing the hips, and then extending them again). Using a rigid tape measure, a measurement was taken from the anterior superior iliac spine to the inferior edge of the medial malleolus on each limb. The leg length difference was determined by subtracting the length of the shorter leg length from that of the longer leg (McCaw, 1992). Intra-examiner reliability for measurement of leg length discrepancy has been

reported as 0.78, while inter-examiner reliability has been reported as 0.80 (Terry et al., 2005).

4.2.3 Statistical Analysis

In this study different methods of statistical analysis were used according to the different types of variables that were investigated (nominal, ordinal or ratio).

1. The Fisher exact test was used to compare the data between uninjured and injured participants for frequency of participants with previous injury, use of orthotics, and using cross-training, stretching and warm-up.

2. Type of previous injury was compared between groups using Chi square test.

3. The level of competitiveness between groups as analysed with a Mann-Whitney test

4. A Friedman's non parametric analysis of variance (ANOVA) test was used to detect differences in running on different surfaces between groups.

5. The unpaired t –test was used to analyse difference in years of training, number of running shoes, kilometers run on each pair of running shoes, frequency and time for stretching, duration of warm-up, average weekly distance, sit and reach test and body composition.

6. The relationship between lower limb alignment and the incidence of injury was evaluated using Pearson product moment correlation and calculating 95% confidence interval for the difference between the mean measures in the injured and non-injured runners. The following comparisons were performed: 1) left side injured group vs. left side uninjured group ; 2) right side injured group vs. right side uninjured group; 3) The afflicted side of the injured group vs. a random side of the uninjured group which was matched to a equal percentage of left or right afflicted side of the injured group.

For all the statistical analysis, the level of significance was accepted as $p \leq 0.05$.

4.3 Results

4.3.1 History of training and injuries

4.3.1.1 Severity of injury (Grade)

The severity of the injury in the runners with Achilles tendinopathy was grade II (pain during running but not restricting running) in 88% of the runners, while 12% reported a Grade I injury (pain only after running). This finding is in keeping with the recruitment strategy, which was to recruit runners with mild to moderate pain so that they would be able to run on a laboratory treadmill to study other parameters described in Chapter 8 Study 5: *The Effect of Pain Development During Running on Lower Limb Kinetic, Kinematic and Muscle Activity Variables in Runners with Achilles Tendinopathy*.

4.3.1.2. Self reported cause of Achilles tendinopathy

The self reported causes of Achilles tendinopathy are depicted in Table 4.1. An increase in training intensity, followed by increase in hill training was the most frequently self reported causes of Achilles tendinopathy in this group of runners.

Table 4.1. Self reported causes of Achilles tendinopathy in runners (%).

Self reported cause of injury	%
Increase in training intensity	33
Increased hill running	19
Increased in training volume	14
Running on a soft surface	8
Running on a hard surface	6
Change in running shoes	3
Others	17

4.3.1.3. Past history of injury

A history of any past running injury was reported by 76% runners in the injured group (Achilles tendinopathy) while 44% of the uninjured runners reported a past history of

any past running injury (p = 0.0187, Fisher exact test). The most common type of previous injury in the injured runners was Achilles tendinopathy, while runners in the uninjured group reported a high prevalence of the iliotibial band friction syndrome (Table 4.2).

Table 4.2. Types of self reported past injuries in the injured and uninjured groups (%).

	Uninjured (N=34)	Injured (N=21)
Patellofemoral pain syndrome	16.7	25.0
Medial tibial stress syndrome	5.6	5.0
Achilles Tendinopathy*	5.6	35.0
Iliotibial band friction syndrome *	44.4	15.0
Plantar fasciitis	0.0	0.0
Others	27.8	20.0

* $p < 0.001$ (Chi Square test, between groups).

4.3.1.4. Training history

Injured runners had ran for significantly more years than the uninjured runners (Uninjured: 8.9 ± 6.0 years; Injured: 14.7 ± 10.1 years; p = 0.0076). However, the competitive level between the two groups was the same (Table 4.3). Most runners in both groups were recreational or recreational/competitive runners.

Table 4.3. Competitive level of runners in the injured and uninjured groups (%).

	Uninjured (N=34)	Injured (N=21)
Elite	4	3
Competitive club level	8	20
Recreational/competitive	55	57
Recreational only	33	20

Both the injured and uninjured runners ran significantly more often on the road than on the track, trail, grass or sand (Table 4.4) and there were no differences in the usual training surfaces between the two groups.

Table 4.4. Usual training surfaces in the injured and uninjured groups (%) (Values are depicted as mean ± SD).

Training surface	Uninjured (N=34)	Injured (N=21)
Road	75.6 ± 28.9*	81.1 ± 19.3**
Track	6.5 ± 21.7	2.8 ± 7.7
Trail	15.2 ± 22.9	12.7 ± 13.0
Grass	1.5 ± 3.2	1.6 ± 4.8
Sand	1.1 ± 4.3	2.1 ± 8.6

* Friedman's test comparing the running surface of injured runners. Road significantly higher than track, trail, grass and sand, $p<0.0001$.

** Friedman's test comparing the running surface of uninjured runners. Road significantly higher than track, trail, grass and sand, $p<0.0001$.

4.3.1.5. Use of footwear (shoes and orthotics)

The reported numbers of running shoes that injured and uninjured runners currently run with were similar in both groups (Uninjured: 1.4 ± 0.6 shoes; Injured: 1.4 ± 0.7 shoes). Similarly, there were no differences in the average kilometers run per running shoe in the injured and uninjured runners (Uninjured: 1442 ± 1000 km; Injured: 1121 ± 819 km). However, a higher proportion of injured runners use orthotics when compared to uninjured runners (Uninjured: 9%; Injured: 48%; $p = 0.0009$, Fisher exact test).

4.3.1.6. Cross training, stretching and warm-up

The self reported history of cross training, stretching habits and warm-up habits of the runners in the injured and uninjured groups is depicted in Table 4.5. A similar proportion of injured and uninjured runners reported engaging in cross training, and the stretching and warm-up habits of the runners in both groups were also similar (Table 4.5). Furthermore, the average weekly running distance was similar between the two groups (Uninjured: 46 ± 19.0 km; Injured: 37 ± 19 km).

Table 4.5. Cross training, stretching and warm-up.

	Uninjured (N=34)	Injured (N=21)
Cross Training (%)	64.7	72.0
Stretching (%)	65.7%	84%
Frequency of stretching (days per week)	2.3 ± 2.0	3.2 ± 1.9
Time of stretching (min)	7.9 ± 15.3	10.1 ± 8.8
Warming –up	61.8%	80.0%
Duration Warm-up (min)	6.7 ± 6.1	4.7 ± 5.0

4.3.2 Body composition and flexibility

The body fat percentage (%) was similar between the two groups (Uninjured: 21.7 ± 5.5 %; Injured: 22.1 ± 5.0 %). However, runners in the uninjured group had significantly higher scores (cm) in the sit-and–reach flexibility test compared with runners in the injured group (Uninjured: 27.5 ± 8.0 cm; Injured: 21.6 ± 10.0 cm; $p = 0.0173$, unpaired t-test).

4.3.3 Lower limb alignment variables

The lower limb alignment variables of the injured limb of the injured group compared with the lower limb alignment variables of the right and left legs of the uninjured group are depicted in Tables 4.6a and Table 4.6b respectively. There were no significant differences between the groups when the right leg was compared between the two groups (Table 4.6a), but left leg subtalar joint passive range of motion for eversion and knee range of motion were significantly different between the groups when the left leg was compared between the two groups (Table 4.6b).

Table 4.6a. Lower limb alignment variables of the right leg in the injured and uninjured groups (Values are depicted as mean ± SD and R values as well as 95% CI are reported).

Right	Uninjured (N=34)	Injured (N=21)	Pearson r	95% CI
Rearfoot alignment (°)	2.4 ± 1.8	1.6 ± 3.0	-0.09	-0.34 to 0.17
Pronation angle at 45 ° (°)	4.6 ± 2.7	5.2 ± 3.3	0.10	-0.16 to 0.35
Q angle (°)	10.1 ± 2.5	9.7 ± 2.4	-0.10	-0.34 to 0.16
Standing foot angle (°)	134.8 ± 10.4	135.7 ± 7.5	0.028	-0.23 to 0.28
Navicular drop (cm)	0.5 ± 0.2	0.6 ± 0.9	0.16	-0.10 to 0.40
Forefoot alignment (cm)	3.2 ± 3.1	2.7 ± 2.9	-0.02	-0.28 to 0.23
Subtalar joint, passive range of motion (inversion) (°)	2.8 ± 2.1	3.3 ± 2.0	0.16	-0.09 to 0.40
Subtalar joint, passive range of motion (eversion) (°)	7.1 ± 4.4	5.2 ± 2.6	-0.23	-0.46 to 0.02
Knee range of motion (°)	140.4 ± 7.6	138.5 ± 7.3	-0.13	-0.37 to 0.13

Table 4.6b. Lower limb alignment variables of the left leg in the injured and uninjured groups (Values are depicted as mean ± SD and R values as well as 95% CI are reported).

Left	Unijured (N=34)	Injured (N=21)	Pearson r	95% CI
Rearfoot alignment (°)	2.8 ± 2.5	1.6 ± 3.4	-0.19	-0.43 to 0.07
Pronation angle at 45 ° (°)	6.1 ± 3.6	5.6 ± 3.4	-0.06	-0.32 to 0.19
Q angle (°)	8.7 ± 1.9	8.9 ± 2.0	0.04	-0.21 to 0.29
Standing foot angle (°)	134.2 ± 8.7	132.4 ± 9.3	-0.10	-0.35 to 0.16
Navicular drop (cm)	0.5 ± 0.2	0.7 ± 1.1	0.15	-0.11 to 0.39
Forefoot alignment (cm)	2.3 ± 2.2	2.3 ± 1.7	0.00	-0.25 to 0.26
Subtalar joint, passive range of motion (inversion) (°)	2.5 ± 1.8	3.1 ± 1.6	0.17	-0.09 to 0.41
Subtalar joint, passive range of motion (eversion) (°) *	8.7 ± 5.5	6.0 ± 3.6	-0.26	-0.49 to 0.00
Knee range of motion (°) **	140.1 ± 8.1	135.6 ± 5.8	-0.29	-0.51 to -0.04

* Significant discriminator between groups, p = 0.043

** Significant discriminator between groups, p = 0.023

The lower limb alignment variables of the injured limb of the injured group compared with the matched limb in the uninjured group is depicted in Table 4.6c. There were no significant differences between the groups for any of the variables.

Table 4.6c. Lower limb alignment variables of the injured limb in the injured group and the matched leg of the uninjured group (Values are depicted as mean ± SD and R values as well as 95% CI are reported).

	Uninjured (N=34)	Injured (N=21)	Pearson r	95% CI
Rearfoot alignment (°)	2.1 ± 2.1	1.6 ± 3.7	0.49	-0.34 to 0.17
Pronation angle at 45 ° (°)	4.8 ± 2.9	5.5 ± 3.3	0.44	-0.16 to 0.35
Q angle (°)	9.8 ± 2.5	9.3 ± 2.4	0.46	-0.34 to 0.16
Standing foot angle (°)	134.5 ± 9.7	135.0 ± 8.8	0.83	-0.23 to 0.28
Navicular drop (cm)	0.4 ± 0.2	0.7 ± 1.2	0.23	-0.10 to 0.40
Forefoot alignment (cm)	2.6 ± 2.8	2.5 ± 2.6	0.85	-0.28 to 0.23
Subtalar joint, passive range of motion (inversion) (°)	2.7 ± 2.2	3.4 ± 2.1	0.22	-0.10 to 0.40
Subtalar joint, passive range of motion (eversion) (°)	7.5 ± 4.6	5.5 ± 2.9	0.08	-0.46 to 0.02
Knee range of motion (°)	140.4 ± 7.0	138.5 ± 7.4	0.32	-0.37 to 0.13

4.3.4. Subjective classification of foot types

The frequency (% of participants) of subjective classifications of foot types in the right and left limbs of runners in the injured and uninjured groups is depicted in Table 4.7a. There were no differences in the classification of foot types between the two groups. Most foot types in both groups were classified as normal.

Table 4.7a. Frequency (% of participants) of different subjectively classified foot types in the right and left foot of the runners in the injured and uninjured groups.

Foot type (%)	Uninjured (N=34)	Injured (N=34)	Spearman r	95% CI
Right foot				
Normal	81	83	-0.01	-0.27 to 0.25
Flat	14	9		
Cavus	5	9		
Left foot				
Normal	76	70	0.07	-0.19 to 0.33
Flat	16	17		
Cavus	8	13		

The frequency of subjective classifications of foot types in the injured limb of the injured runner group and the matched limb of the uninjured group is depicted in Table 4.7b. There was no significant difference in the classification of foot types between the two groups. Most foot types in both groups were classified as normal.

Table 4.7b. Frequency (% of participants) of different subjectively classified foot types in the injured limb of the injured group and the matched limb of the uninjured group.

Foot type (%)	Uninjured (N=34)	Injured (N=21)	Spearman r	95% CI
Normal	76	87		
Flat	16	4	-0.12	-0.37 to 0.14
Cavus	8	9		

Finally, leg length discrepancy (cm) was similar between the two groups (Uninjured: 1.0 ± 0.7 cm; Injured: 0.7 ± 5.5 cm, Pearson r = 0.79, 95%CI = -0.22 to 0.29).

4.4 Discussion

The main finding of this study was that injured runners have a higher prevalence of a previous running injury, ran for more years and have reduced flexibility compared to uninjured runners. These findings support the hypothesis that specific intrinsic and extrinsic factors are associated with Achilles tendinopathy, and partially support the findings from the literature review in Chapter 2.

There is strong evidence that a history of a previous running injury is an intrinsic risk factor for a running injury (Walter et al., 1989; Macera et al., 1989; Taunton et al., 2003; van Gent et al., 2007). However, this has not been reported in runners with Achilles tendinopathy, hence this is a novel finding from this study. The reasons for this association between a history of a previous running injury and Achilles tendinopathy is not clear. A number of factors could be responsible for this observation.

Firstly, a history of a past running injury may result in mild discomfort or pain during running. The development of discomfort or pain during running may alter lower limb biomechanics which can then alter load distribution and result in subsequent injury. Secondly, incomplete healing of a previous injury could increase the risk of subsequent injury. In this study, Achilles tendinopathy was a recurrent injury in 35% of the injured runners (Table 4.2). Finally, runners with a past history of injury may have a genetic predisposition to injury as recently published studies have reported (Mokone et al., 2006; September et al., 2008) which may explain the recurrence of the injury.

In this study, runners with Achilles tendinopathy reported a longer history of running compared with uninjured runners. It has been reported that less running experience is associated with an increased risk of all running injuries (Macera et al., 1989; Satterthwaite et al., 1999; Taunton et al., 2002), supporting this finding other study showed that running for more years is a protective mechanism against injury (Fredericson and Misra, 2007). However, data are not consistent, and this association may vary according to injury type (van Gent et al., 2007). Furthermore, there are some studies that report no association between running years and injury risk (Walter

et al., 1989). As far as is known, there is only one study where the relationship between years of running and Achilles Tendinopathy was investigated (McCrory et al., 1999). The findings from this study are similar to the results reported in this study namely that increased years of running are associated with Achilles tendinopathy in runners. The association between more years of running and Achilles tendinopathy is expected, as this is a degenerative injury, and it may take longer for the manifestation of the symptoms. However, the recruitment of participants which have not been injured during the last 2 years may have promoted some selection bias, which may have influenced these results.

Another aetiological factor in this study that was associated with this injury was reduced flexibility (as shown by the sit and reach test) in injured runners. Achilles tendinopathy has been associated with reduced flexibility of the gastrocnemius and soleus muscles measured by range of plantar and dorsiflexion in a retrospective study (Clement et al., 1984). The present study did not measure the range of plantar and dorsiflexion, however the sit and reach test is also an indirect measure of the flexibility of the gastrocnemius. Previous studies have associated the reduced flexibility of the gastrocnemius with the older age seen in Achilles tendinopathy runners (Clement et al., 1984; Kannus et al., 1989; Alfredson and Lorentzon, 2000). However, in this study age was not significantly different between groups (Chapter 3: *Research Methodology*).

In this study, the number of runners who stretch, the frequency of stretching per week and the duration of stretching was similar between the two groups (Table 4.5). However, in contrast with this findings McCrory *et al.* (1999), found that Achilles injured runners were less likely to incorporate stretching in their daily routine. Despite similar reported stretching habits between the two groups seen in this present study, general flexibility was decreased in the injured group. This observation may be because although duration and frequency of stretching were measured more information about the quality of stretching (e.g. way that exercise was conducted and type of stretching exercises) were not recorded and maybe differed between the groups.

In this study, 33% of the injured runners associated the cause of their injury with increased intensity of training (Table 4.1). It has been reported in several studies that training error are associated with Achilles Tendinopathy (Clement et al., 1984; McCrory et al., 1999; Milgrom et al., 2003; Barr and Harrast, 2005). Other study have found that Achilles injured runners tended to train at a faster running pace compared with controls (McCrory et al., 1999). The present study did not assess running pace, although a similar percentage of runners in both groups classified their running as either competitive or recreational (Table 4.3). In one study, an increased running intensity has been associated with 6% of the running injuries; while a sudden increase in training volume was more likely to be associate with Achilles running injury (13% of the self-reported causes) (Clement et al., 1984). As presented in Table 4.1, a self-reported increased training volume was the third most frequent cause of Achilles tendinopathy after increased intensity and hill training, but average weekly training distance was similar in both groups. It must be noted that the average training distance was only reported from the last three months of training, at the time when the injured runners were already suffering from the injury. Therefore, the relationship between risk of Achilles tendinopathy and increased training volume or intensity is inconclusive. Further prospective studies should be conducted to assess this relationship, and it would be important to record training volume and intensity very accurately.

Another training error commonly associated with Achilles tendinopathy is limited warm-up (Milgrom et al., 2003; Barr and Harrast, 2005). In one study in military recruits it was shown that the incidence of Achilles injury is more frequent in the winter than in the summer (Milgrom et al., 2003), and this may be related to a limited warm-up. In the current study, the number of runners who reported performing a warm-up in both groups as well as the duration of the warm-up was similar (Table 4.5). Therefore these results do not support the hypothesis that a reduced warm-up is associated with an increased risk of injury. The data should however be interpreted in the context that it was self-reported data.

Cross training is another factor that has been associated with a reduced risk of running injuries (Clement et al., 1981; Walter et al., 1989; Taunton et al., 2003). The basis for a possible reduction in the risk of injuries with cross training has its origins in the idea

that cross training is associated with reduced impact forces during running and because adaptations occurred after performing other non-weight bearing activities (e.g. cycling, swimming,). However, in the present study (Table 4.5) and in support with the results of other studies (Clement et al., 1981; Walter et al., 1989; Taunton et al., 2003) this argument does not hold and there is no evidence that cross training reduces injury risk in runners.

Running on hard surfaces has been suggested by some (Macera et al., 1989; Messier et al., 1991) but not others (Jacobs and Berson, 1986; van Mechelen, 1992) as a risk factor for running injuries. Reports are not consistent and also vary according to gender (Macera et al., 1989). In the present study, both groups of runners frequently ran on the road rather than on other surfaces, and there were no statistical differences between the choices of running surfaces between the two groups (Table 4.4).

Running with old running shoes (Wen et al., 1997; Taunton et al., 2003) and using increased numbers of running shoes (Wen et al., 1997) have both been associated with an increased risk of running injuries. The rationale is that the reduced impact absorption of the old running shoe may increase the risk of injury. However, there is limited evidence from the literature to support this and the effect may also vary according to gender (Taunton et al., 2003). Most studies are limited by the fact that multiple injuries rather than a single injury were examined. The results on the current study show that there were no differences between the two groups for the reported number of running shoes, and the average kilometres run per shoe (Section 4.3.1.5. *Use of footwear (shoes and orthotics)*). These data suggest that shoe age is not associated with the development of Achilles tendinopathy.

Control of pronation is another factor that has been associated with running injury. The present study found that the use of orthotics was significantly higher in the injured group compared with the uninjured group (Section 4.3.1.5. *Use of footwear (shoes and orthotics)*). It is likely that this observation is because injured runners use orthotics to treat their injury as orthotics have been used as a treatment modality for a number of running injuries, including Achilles tendinopathy (D'Ambrosia, 1985; Blake and Denton, 1985; Gross et al., 1991). Although, in a more recent systematic

review the use of orthotics to treat running injuries has been questioned (Razeghi and Batt, 2000)

Anthropometric measurements such as body weight, height and BMI have also been linked to the risk of developing running injuries (Walter et al., 1989; Macera et al., 1989; Taunton et al., 2002). The underlying hypotheses are 1) that a reduced lean body mass would result in a lack of ability to compensate for the impact forces involved during running (Neely, 1998; Taunton et al., 2002), or 2) that an increased BMI or percent body fat could predispose to injury because the impact forces would be greater. In most reports height, weight and body mass index were studied but no studies have reported on the possible relationship between percent body fat and injury risk. In the present study percentage of body fat and consequently lean body mass were similar between injured and uninjured groups (Section 4.3.2 *Body composition and flexibility*), thereby not supporting the hypothesis that percent body fat is related to running injuries.

The relationship between height and running injury risk has also received some attention. Previous studies have shown contradictory results. In one study, taller runners were more prone to injury (Walter et al., 1989) while in another study shorter runners who were more prone to injury (Taunton et al., 2002). The same contradictory results were found for body mass and body mass index (BMI), showing an increase in risk of injury with increased body mass and BMI (Taunton et al., 2002; Taunton et al., 2003), while another study has not shown an association between BMI and injury (Walter et al., 1989). In the present study height and body mass were significantly higher in the injured group (Table 3.1 - Chapter 3: *Research Methodology*), supporting the fact that an increase in body mass and height may be associated with injury. Therefore, it can be concluded that increased body mass and height might be associated with injury but there is no association between lean body mass and injury as previously presented.

In a number of reports, static lower limb alignment variables have been associated with running injury risk (McKenzie et al., 1985; Lysholm and Wiklander, 1987; Brunet et al., 1990; McCaw, 1992; Kvist, 1994; Wen et al., 1997; Lun et al., 2004). The rationale is that skeletal misalignment may result in adverse biomechanics, which

may cause injury. In the present study conventional clinical lower limb alignment measurements were compared between runners with Achilles tendinopathy and uninjured runners (injured limb in the Achilles tendinopathy group compared with both the same limb and a matched limb of the uninjured group).

The only variables that were found to be different between the two groups were knee range of motion and subtalar joint passive range of motion for eversion for the left limb (Table 4.6a), these values were significantly lower in the injured group. The reduced knee range of motion of the injured runners may be related to the reduced hamstring flexibility seen on the sit and reach test. The result may also be associated with the reduced range of motion of knee flexion during running which were observed in Chapter 5. Study 2: *Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*. This finding however, will not be discussed in detail now, but this will be explored further in Chapter 5.

The reduced subtalar ankle mobility, which was observed in the Achilles tendinopathy group in this present study (Table 4.6a) has been observed previously (Kvist, 1994). It has been documented that 60% of runners with Achilles tendinopathy had reduced subtalar and ankle joint mobility. However, in the study of Kvist (1994) there was no control group. The observed association between reduced knee and subtalar range of motion and Achilles tendinopathy needs to be interpreted with caution as no association was found for the same measurements for the right limb and the injured limb against matched limb.

Another anatomical measurement that is reported to be associated with running injuries is foot type (Messier et al., 1991; Kvist, 1994). Although cavus arch has been associated with Achilles tendinopathy (Kvist, 1994; McCrory et al., 1999), this was not supported by the finding of this study ((Tables 4.7a and 4.7b), which is similar to those observed in three prospective studies (Montgomery et al., 1989; Wen et al., 1997; Lun et al., 2004).

Forefoot alignment has been associated with Achilles tendinopathy (Clement et al., 1984; Kvist, 1994), however, no such association was found in this present study

(Tables 4.6a, 4.6b and 4.6c). Similarly, leg length discrepancy has been associated with running injuries (Brunet et al., 1990; McCaw, 1992) (Gross, 1983; Messier et al., 1991), however similar association was not found in this study.

Finally, a number of other lower limb alignment measurements including rearfoot alignment, pronation angle at 45°, Q angle, standing foot angle and navicular drop have all been reported as possible risk factors that can be associated with running injuries in general (Lysholm and Wiklander, 1987; Messier et al., 1991; Wen et al., 1997; Hintermann and Nigg, 1998; Cornwall and McPoil, 1999). However, the present study does not confirm the association between these variables and Achilles tendinopathy in runners (Tables 4.6a, 4.6b and 4.6c) and these are supported by the results from a more recent prospective study where no association between lower limb alignment and the incidence of injury for most of the injuries (excluding patellofemoral pain) was documented (Lun et al., 2004).

It is important to point out that there are inherent limitations in this study. Firstly, the reliability (intra-examiner and inter-examiner) of the clinical lower limb measurements that were conducted is not always high enough to confidently use the tests. This is a common criticism of many clinical measurements and is acknowledged. However, the data on intra-examiner and inter-examiner reliability of tests was reported, and results have to be interpreted with this in mind. Intra and inter-reliability testing were not repeated in the present study and this can also be criticized. However, the examiner was well trained by an experienced podiatrist and performed a series of pilot testing. A second limitation of this study was that the questionnaire data was self-reported which introduces recall baias, and reflects the participant's own opinion and description. Thirdly, in this study, dorsi-plantarflexion range of motion was not measured, and this can be considered a limitation of the study. Previous studies have noted an association between Achilles tendinopathy and this varaible (Kvist, 1994). Additionaly as this is a case-control study design, it is not possible to link factor causally to the injury, hence only an association between the injury and risk factors can be suggested. Prospective cohort studies or randomized controlled intervention studies are needed to determine a cause-effect relationship between the variables and injury risk. This limitation also applies to the other experimental chapters in this thesis where other risk factors that may be associated with Achilles

tendinopathy will be explored further. Finally, there was a large variation on the length of injury from these participants (4 - 121 months). This large variation might have implications on the results of this study, in particular in relation to flexibility and lower limb alignment measurements as well as kinetics, kinematics and muscle activity responses of the injured runners (Chapters 5 to 8).

In conclusion, this case-control study in runners with Achilles Tendinopathy showed that runners with Achilles tendinopathy had a higher prevalence of a history of a previous running injury, which may be related to a number of factors including biomechanics alteration due to pain, incomplete healing and genetic predisposition. Furthermore, the result of this study showed that runners with Achilles tendinopathy tend to run for more years, have an increased weight and height, and had reduced flexibility of the posterior leg and trunk muscles measured by the sit and reach test.

Therefore, the results of this study show that some extrinsic (years of running) and intrinsic (previous injury, weight, height and flexibility) risk factors appear to be associated with Achilles tendinopathy. Importantly, it was also shown that lower limb biomechanical alignment parameters which are commonly listed as risk factors for Achilles tendinopathy (Krivickas, 1997), were not associated with this injury. Therefore, other possible risk factors for Achilles tendinopathy need to be identified. In the next two studies, biomechanical factors, specifically kinematic, kinetic and muscle activity parameters as well as variability in these parameters, as possible risk factors for Achilles tendinopathy will be investigated.

CHAPTER 5. STUDY 2: LOWER LIMB KINETIC, KINEMATIC AND MUSCLE ACTIVITY VARIABLES AS RISK FACTORS ASSOCIATED WITH ACHILLES TENDINOPATHY IN RUNNERS

5.1 Introduction

In the previous Chapter, static lower limb alignment variables as possible risk factors that may be associated with Achilles tendinopathy were studied. It was concluded that very few of the postulated lower limb alignment variables, as measured by clinical assessment, were in fact associated with Achilles tendinopathy. Therefore, a more detailed analysis of other dynamic biomechanical parameters as possible factors that may be associated with Achilles tendinopathy is warranted.

Although Achilles tendinopathy is one of the most common injuries incurred by distance runners (Smart et al., 1980), it is important to note that the scientific evidence for the majority of the extrinsic and intrinsic risk factors associated with Achilles tendinopathy is based on clinical assessment rather than evidence derived from well conducted experimental studies in a laboratory setting (Smart et al., 1980; Kvist, 1994; Jones, 1998; Kader et al., 2002; September et al., 2007). In general, the association between intrinsic dynamic biomechanical factors and the risk of Achilles tendinopathy in runners has not received much attention in the literature.

In only one case-control study, muscle strength and lower limb kinematic and kinetic variables were studied in 31 runners with Achilles tendinopathy and 58 uninjured controls. The findings of this study showed that Achilles tendinopathy was associated with greater plantar and dorsiflexion torque during a isokinetic test and an earlier and increased peak pronation (McCrory et al., 1999). However, the authors did not find any relationship between the kinetic variables and Achilles tendinopathy. The relationship between lower limb kinetic and kinematic variables and Achilles tendinopathy therefore still needs further investigation. Furthermore, in recent years, the role of impact force and foot pronation as factors that increase running injury risk has been questioned. (Nigg, 2001). Rather, the importance of neuromuscular control

during running has been suggested as an important variable to investigate in the aetiology of running injuries.

In particular, it has been speculated that the increase in muscle activity before and after heel strike is important to reduce muscle vibration during impact forces (Nigg and Wakeling, 2001). It has been shown that muscle activity can change according to running shoe (Wakeling et al., 2002b), running surface (Boyer and Nigg, 2006), orthotics (Mundermann et al., 2006), running speed (Kyrolainen et al., 2005) and fatigue (Hanon et al., 2005). However, only one study (van Lent et al., 1994), has investigated the muscle activity patterns during running in injured participants. (van Lent et al., 1994) have compared muscle activity during walking and running between patients with anterior cruciate ligament (ACL) deficiency and uninjured controls. In their study, a reduction in muscle activity during running, and after 10 minutes of walking was reported in the ACL deficient group. However, no studies have investigated muscle activity in runners with Achilles tendinopathy.

Therefore, it is clear that lower limb kinematic and kinetic variables in runners with Achilles tendinopathy has not been studied extensively. Furthermore, the relationship between altered lower limb muscle activity in runners with Achilles tendinopathy has, to our knowledge, not been studied. Therefore, the aim of this study was to investigate the kinetic, kinematic and muscle activity characteristics in runners with Achilles tendinopathy compared with matched uninjured runners. Based on previous literature, it is hypothesized that runners with Achilles tendinopathy would have similar kinetics but altered kinematics and muscle activity compared to uninjured runners.

5.2 Methods

The participant characteristics of the two groups of runners have already been discussed in Chapter 3 *Research Methodology* (Table 3.1). The experimental procedures and data analysis of parameters that will be reported in this study were also described in Chapter 3 *Research Methodology*.

5.2.1 Statistical Analysis

All data were expressed as means ± standard deviation ($X \pm s$). Differences between the two groups were compared using the t test for independent variables. The false discovery procedure was used to account for multiple comparisons (Curran-Everett, 2000). This practical method overcomes some of the pitfalls associated with other common techniques (Bonferroni, Newman-Keuls and least square difference). Statistical significance was accepted as $p \leq 0.05$ before the adjustments for multiple comparisons were made. To measure the magnitude of a treatment effect, the effect size was reported when statistical differences were observed. The Cohen' standard method (d) was the method selected to measure the effect size. The differences were interpreted as; $d = 0.2$, *small effect size*; $d = 0.5$, *medius effect size* and $d = 0.8$ *large effect size* (Cohen, 1988).

5.3 Results

The temporal distance parameters were similar between the injured and uninjured runners (Table 5.1)

Table 5.1. Temporal distance parameters; uninjured vs. injured. (Values are depicted as means ± SD).

	Uninjured (N = 34)	Injured (N = 21)	p value
Speed (m/s)	3.03 ± 0.38	2.96 ± 0.37	0.622
Stride length (m)	2.23 ± 0.28	2.17 ± 0.30	0.488
Stride time (s)	0.74 ± 0.04	0.74 ± 0.06	0.972
Stride frequency (strides/min)	82 ± 5	82 ± 7	0.889
Contact time (s)	0.17±0.01	0.17±0.02	0.966

The kinetic variables were similar between uninjured and injured runners (Table 5.2).

Table 5.2. Kinetic variables; uninjured vs. injured. (Values are depicted as means ± SD).

	Uninjured (N = 34)	Injured (N = 21)	p value
HBF (BW)	0.22 ± 0.05	0.20 ± 0.05	0.426
HPF (BW)	0.16 ± 0.04	0.16 ± 0.04	0.811
VIF (BW)	1.33 ± 0.25	1.45 ± 0.23	0.106
VLR (BW/s)	42.76 ± 9.01	44.79 ± 11.27	0.482
VPF (BW)	2.22 ± 0.16	2.18 ± 0.23	0.444

Abbreviations*: HBF = horizontal braking force; HPF = horizontal propulsive force; VIF = vertical impact force; VLR = vertical loading rate; VPF = vertical propulsive force*

The kinematic variables in the uninjured and injured runners are depicted in Table 5.3. There were no statistical differences in kinematic parameters after correcting with the use of false discovery method. However, there was a tendency (p=0.017) for a decrease in the range of knee flexion between heel strike and midstance (KROM) in the injured runners compared with the uninjured runners (Table 5.3 and Figure 5.1).

Table 5.3. Kinematic variables; uninjured vs. injured (Values are depicted as means ± SD).

	Uninjured (N = 34)	Injured (N = 21)	p value
Hic (°)	39.8 ± 9.5	42.4 ± 7.2	0.326
Hto (°)	2.1 ± 11.9	3.8 ± 5.5	0.564
HROM (°)	36.7 ± 11.6	38.7 ± 7.1	0.500
KswE (°)	15.8 ± 7.8	20.1 ± 7.7	0.067
Kic (°)	15.9 ± 7.4	20.2 ± 7.2	0.049
Kst (°)	41.4 ± 8.5	42.2 ± 4.8	0.722
KROM (°)	25.5 ± 4.6	22.0 ± 5.5	0.017
Asw (°)	-12.7 ± 4.8	-11.5 ± 5.6	0.425
Aic (°)	-11.0 ± 8.6	-11.5 ± 5.8	0.815
Ast (°)	-21.4 ± 9.2	-20.9 ± 3.4	0.828

Abbreviations: *Hic = hip angle at initial contact; Hto = hip angle at toe off; HROM = hip range of motion; KswE = knee angle at terminal swing phase; Kic = knee angle at initial contact; Kst = knee angle at stance phase; KROM = knee range of motion; Asw = ankle angle at terminal swing phase; Aic = ankle angle at initial contact; Ast = ankle angle at stance phase.*

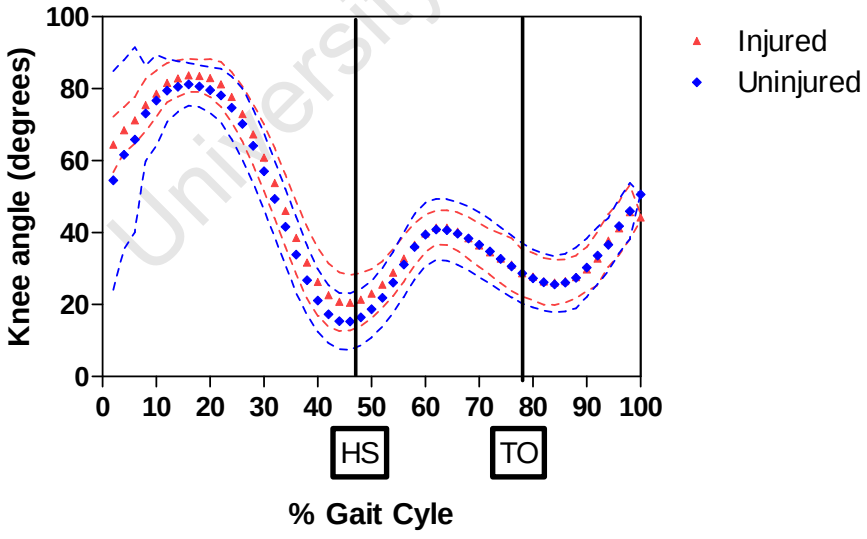

Figure 5.1. The knee sagittal plane kinematics (knee angle in degrees) in the injured and uninjured runners. Values are presented throughout the full gait cycle (0-100%) and variance is displayed as standard deviation.

The IEMG activity 100 ms before (pre) and 100 ms after (post) heel strike of the lower limb muscles is depicted in Table 5.4. There was a significant decrease in the pre-heel strike IEMG activity of TA in the injured runners compared with the uninjured runners (Table 5.4 and Figure 5.2). Likewise, post-heel strike IEMG activity of RF was significantly lower in the injured runners compared with the uninjured runners (Table 5.4 and Figure 5.3).

Table 5.4. IEMG activity (%*s) pre (100 ms before heel strike), post (100 ms after heel strike) uninjured vs. injured runners. (Values are depicted as means ± SD).

	Uninjured (N = 34)	Injured (N = 21)	p value
TApre	22.0 ± 6.0	17.3 ± 6.0	0.007*
TApost	13.0 ± 7.2	12.2 ± 8.0	0.694
BFpre	17.8 ± 4.7	16.1 ± 7.9	0.337
BFpost	17.5 ± 7.6	18.5 ± 7.1	0.625
PEpre	11.3 ± 5.3	10.2 ± 4.5	0.473
PEpost	28.4 ± 8.4	25.3 ± 10.2	0.254
GMpre	12.3 ± 3.1	13.0 ± 5.9	0.624
GMpost	24.3 ± 10.2	18.1 ± 7.9	0.038
LGpre	9.6 ± 4.1	9.6 ± 5.4	0.963
LGpost	27.5 ± 8.2	23.7 ± 10.4	0.153
RFpre	13.1 ± 4.3	14.8 ± 6.5	0.262
RFpost	33.5 ± 8.3	21.6 ± 9.6	0.00002*

The significant comparisons after adjustment with the false recovery procedure are shown as *.

Effect size: TApre $d = 0.78$ i.e. large effect size

RFpos $d = 1.33$ i.e. large effect size.

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

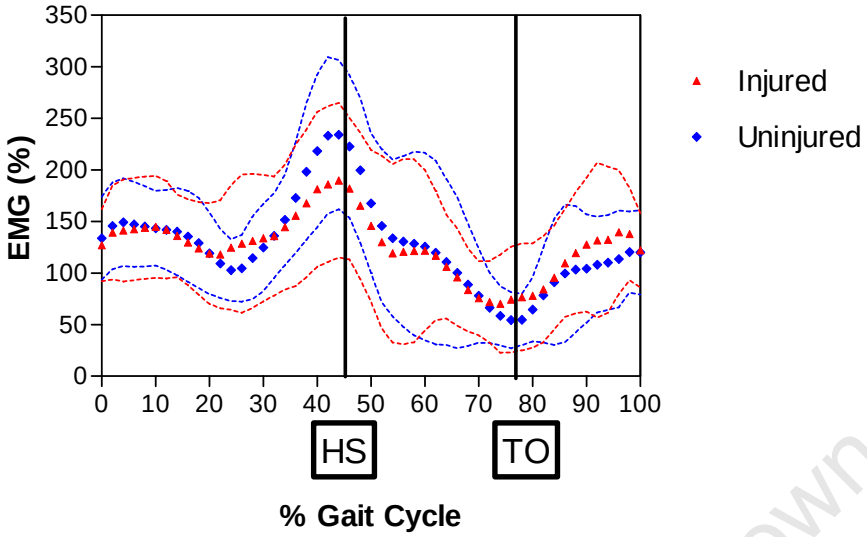

Figure 5.2. The EMG activity (%) of the tibialis anterior (TA) muscle of the injured and uninjured runners. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

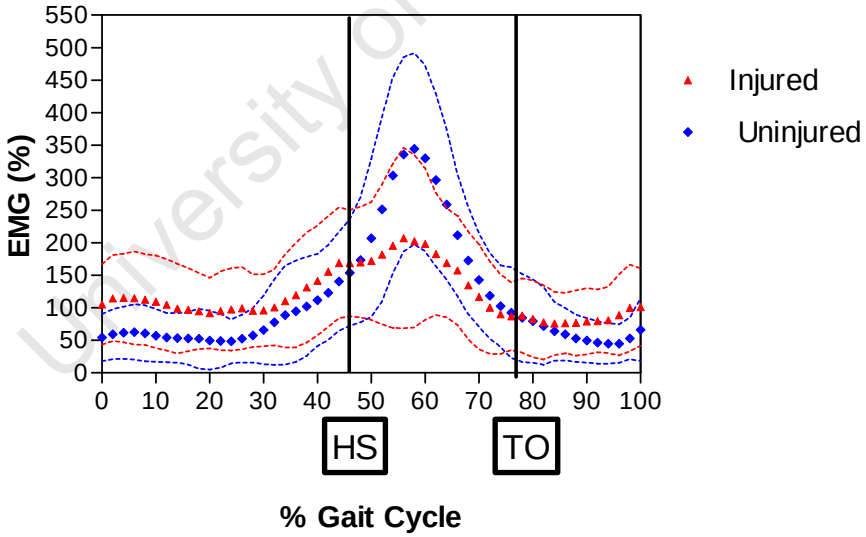

Figure 5.3. EMG activity (%) rectus femoris (RF) of injured and uninjured runners. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

The IEMG activity of the RF during the stance phase and the peak IEMG activity during the same period was significantly lower in the injured runners compared with the uninjured runners (Table 5.5 and 5.6, Figure 5.4). Similarly, the peak IEMG activity of the RF occurred significantly earlier in the injured runners compared with the uninjured runners (Table 5.6, Figure 5.4).

Table 5.5. IEMG activity of the whole gait cycle (GC) and on stance phase (SP) (%*s). (Values are depicted as means ± SD).

	Uninjured (N = 34)	Injured (N = 21)	p value
TA GC	12800 ± 401	12630 ± 777	0.227
TA SP	4272 ± 1571	4216 ± 1797	0.906
BF GC	12900 ± 659	12810 ± 625	0.630
BF SP	5942 ± 1179	6733 ± 1640	0.054
PE GC	13000 ± 490	12960 ± 598	0.763
PE SP	8682 ± 1745	8077 ± 2447	0.319
GM GC	12580 ± 492	12500 ± 648	0.649
GM SP	6700 ± 1649	6173 ± 1271	0.257
LG GC	12950 ± 532	12710 ± 584	0.134
LG SP	8996 ± 1898	8141 ± 2579	0.183
RF GC	12640 ± 441	12639 ± 566	0.978
RF SP	8207 ± 1304	6183 ± 2011	0.0001*

The significant comparisons after adjustment with the false recovery procedure are shown as *.
Effect size RF SP d = 1.19 i.e. large effect size
Abbreviations: GC = gait cycle, SP = stance phase, *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

Table 5.6. Peak EMG activity on the stance phase (PSP) (%) and time of occurrence of peak EMG activity on the stance phase (TSP) (% gait cycle). (Values are depicted as means ± SD).

	Uninjured (N = 34)	Injured (N = 21)	p value
TA PSP (%)	244 ± 81	211 ± 83	0.164
TA TSP (s)	52 ± 10	54 ± 10	0.535
BF PSP (%)	273 ± 82	314 ± 91	0.106
BF TSP (s)	63 ± 7	63 ± 7	0.831
PE PSP (%)	402 ± 93	381 ± 140	0.527
PE TSP (s)	61 ± 6	61 ± 9	0.750
GM PSP (%)	318 ± 126	269 ± 82	0.149
GM TSP (s)	59 ± 6	62 ± 8	0.234
LG PSP (%)	382 ± 115	366 ± 140	0.645
LG TSP (s)	64 ± 6	63 ± 9	0.637
RF PSP (%)	413 ± 94	305 ± 84	0.002*
RF TSP (s)	59 ± 5	54 ± 8	0.003*

The significant comparisons after adjustment with the false recovery procedure are shown as *.
Effect size RF PSP $d = 1.21$ i.e. large effect size
RF TSP $d = 0.74$ i.e. medium effect size
Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

5.4 Discussion

The main novel finding of this study was that runners with Achilles tendinopathy had a reduction in the muscle IEMG activity of the tibialis anterior (TA) and rectus femoris (RF) muscles during different phases of the running cycle compared to the uninjured runners. Therefore, the stated hypothesis can be accepted for similar kinetics but altered muscle activity between injured and uninjured runners.

In the present study vertical impact force peak and loading rate were similar between the two groups (Table 5.2). The same results were found in another study with runners who had Achilles tendon injuries (McCrory et al., 1999). However, in the present

study the muscle activity was significantly lower for TA and RF in the injured runners (Table 5.4). Furthermore, knee kinematics during contact (Table 5.3) tended (significant before correction for multiple comparisons) to be different between the two groups. This suggests that the similar impact force was not associated with a similar pattern of muscle activity and kinematics adjustments in injured compared with the uninjured runners.

According to the impact force and muscle tuning paradigm (Nigg, 2001), impact force is sensed by mechanoreceptors in the lower extremity, and the information is then transmitted to the central nervous system, which then results in the dynamic response and the movement adjustment. It could, therefore be speculated that the similar impact forces and reduced muscle activity observed in the present study indicate that injured runners do not adjust their muscle activity to impact forces in the same way as uninjured runners, possibly due to an altered in sensitivity of the mechanoreceptors mechanism (afferent pathway) or to a restriction on transferring the signal information by the central nervous system to the muscle groups (efferent pathway). Muscle activity and plantar pressure were significantly affected when sensory feedback on the sole of the foot was altered by cooling with ice (Nurse and Nigg, 2001), showing that mechanoreceptors on the sole of the foot are important to determine gait patterns. In accordance with this, it could also be hypothesized that the mechanoreceptors in the Achilles tendon could possibly be damaged due to injury and, therefore, the sensory input may have been affected.

In the present study, the injured group tended (significant before correction, for multiple comparisons, $p = 0.049$) to have a higher knee flexion angle at initial contact (Table 5.3, Figure 5.1). According to other studies, the increase in knee flexion at contact reduces the axial stiffness of the body (Lafortune et al., 1996a) but increase the shock traveling throughout the shank (Lafortune et al., 1996b). Additionally, the injured group tended (significant before correction, $p=0.0017$) to have a lower range of motion of knee flexion (Table 5.3, Figure 5.1). Thus, the injured runners seem to run in a more flexed posture, compared with the uninjured runners. Running with the knee in a more flexed position (Groucho running) can reduce the vertical stiffness but it would increase the energy cost (McMahon et al., 1987). In addition, the tendency to reduce ROM of knee flexion in the injured group during the stance phase (Table 5.3),

could indicate a weakness of the quadriceps during eccentric contraction (O'Connor et al., 2001). This finding is supported by the observed lower IEMG activity of the RF during the stance phase observed in this group (Table 5.5 and 5.6 and Figure 5.3).

It has been suggested reduced gastrocnemius flexibility may increase knee flexion and foot pronation during walking or running, which may then promote injury (Clement et al., 1984). However, the present study results have found the opposite, as there was a tendency ($p=0.017$, significant before correction) to reduce the range of motion of knee flexion during running in the uninjured runners, thus, not supporting this hypothesis. Therefore it is suggested that reduced flexibility, as previously shown with the sit and reach test (Chapter 4. Study 1. *Training, Flexibility and Lower Limb Alignment Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*), may promote a reduced knee flexion during running by reducing their range of motion.

As far as is known, this present study is the first study to report that altered muscle activity of the lower extremity muscles in Achilles tendinopathy runners is associated with a lower limb overuse injury. In the past, lower limb biomechanical analysis was limited to the assessment of only the kinematic and kinetic variables (Clement et al., 1984; McCrory et al., 1999; Hreljac et al., 2000; Milner et al., 2006). However, in recent years, there has been a shift in focus, and the possible role of muscle activity in the development of running related injuries has been identified. Muscle activity can affect different aspects of the gait cycle, such as joint angle and velocity, joint stiffness, vibration of soft tissues, joint loading and stability (Nigg and Wakeling, 2001). It was hypothesized that the impact forces during the ground contact phase are important to promote alterations in muscle activity before (pre-heel strike) and during ground contact and this would reduce soft tissue vibration and therefore minimize joint and tendon loading (Nigg, 2001; Nigg and Wakeling, 2001).

In this study, muscle activity was investigated in the period of 100 ms before and after heel strike (Table 5.4). Muscle activity before heel strike can be interpreted as an important mechanism to protect the body against the shock of landing, which occurs at the time of the heel strike. It is assumed that pre-activation of the muscles is important to increase the sensitivity of the muscle spindle. This leads to an

amplification of the stretch reflex which then increases the muscle activity of the knee extensors and the plantarflexors thereby enhancing the stiffness of the tendon muscular system to absorb the impact (Komi, 2000). It is presumed that the muscle activity is predetermined through impact signals from the previous landing of the foot (Nigg and Wakeling, 2001). Several studies have measured muscle activity before heel strike in a window period of between 100 to 150 ms before heel strike. These studies have found that muscle pre-activation is affected by running speed (Kyrolainen et al., 2005), shoe material (Nigg et al., 2003) and the level of running proficiency (Paavolainen et al., 1999).

Considering that the main role of muscle pre-activity is to protect and prepare the muscles for landing, the reduction of muscle pre-activity of TA in the injured group (Figure 5.2), could indicate a reduction in muscle stiffness and an increase in joint loading. Additionally, there was no alteration on ankle kinematics in this study (Table 5.3), showing that the reduction in muscle activity did not alter the skeletal position.

The tibialis anterior muscle is active during the swing phase to control plantarflexion and to initiate dorsiflexion. During the initial stance phase, this muscle acts eccentrically and is an important muscle for ankle stabilization (McClay et al., 1990). In the present study, the reduction in muscle activity of TA was only observed before heel strike, which could influence the muscle force after heel strike as there is a delay of about 50 ms between the onset of EMG activity and the development of muscle force (Novacheck, 1998b).

A study which investigated TA activity when the participants were running either barefoot or wearing shoes (von Tscharner et al., 2003), showed that TA was more active before heel strike than after heel strike in both conditions. Similar results were found in the present study, but the range between pre and post heel strike was larger in the uninjured group than in the injured group (Uninjured TA pre: 22.0 ± 6.0%*s; TA post: 13.0 ± 7.2 %*s, range of 9.0 %*s; Injured: TA pre: 17.4 ± 6.0 %*s; TA post: 12.2 ± 8.0 %*s, range of 5.2%*s). In the previously mentioned study it was also found that the intensity of pre heel strike TA muscle activity increased compared to post heel strike when wearing shoes, suggesting that TA is adjusted to exterior conditions (von Tscharner et al., 2003). The reduced range between of TA pre and post IEMG

activity in the injured population supports the previous assumption that injured runners are less capable to adjust to exterior conditions.

During the contact phase, the main role of muscle activity is to alter skeletal position and velocity (Nigg and Wakeling, 2001). The results from this study showed that there was an alteration in the muscle activity and and a tendency to associated changes in kinematics in the stance phase in the injured runners (Table 5.3, 5.4 and 5.5). The RF seems to be the most affected muscle during the running cycle. The results from the present study showed that RF activity was reduced during the first 100 ms and during the whole stance phase period in the injured group (Table 5.4 and 5.5 and Figure 5.1). Additionally, the peak IEMG activity of this muscle was lower and occurred earlier in the group with Achilles tendinopathy (Table 5.6). The RF is mainly active during the late swing phase and early stance phase and is relatively inactive during the mid-stance to the toe-off phase (McClay et al., 1990). RF acts eccentrically during the early stance phase to restrain the movement of the tibia as the knee flexes and it is an important muscle for absorbing impact during the stance phase (Novacheck, 1998b). The reduced IEMG activity of this muscle in the injured runners may indicate less control of the tibia at early stance and a decrease in shock absorption, which could be a mechanism for injury.

According to Novacheck, (1998) tissues such as the Achilles tendon, plantar fascia, quadriceps and hip abductors dissipate force over the first half of the stance phase. Therefore, it can be hypothesized from the result of the present study that the reduction in muscle activity of the RF could perhaps increase the force in other soft tissues such as the Achilles tendon and plantar fascia or even in other quadriceps muscles that were not analysed in this study.

The gluteus medius is an important muscle, which stabilizes the hip with respect to the thigh during early stance phase. The GM is mainly active on late swing phase and early stance (McClay et al., 1990). In this study (Table 5.4), the EMG activity of GM 100 ms after heel strike tended (significant before correction for multiple comparisons, $p = 0.038$) to be lower in the injured compared to the uninjured runners. Weak hip abductors have been associated with the iliotibial band syndrome (Fredericson et al., 2000). The relationship between weak GM muscles and Achilles

tendinopathy is not clear, but a weak GM muscle could cause femoral adduction and internal rotation of the knee and the tibia would then rotate internally, possibly increasing pronation. However, this finding should not be over interpreted as there were no statistical differences between the groups.

It is estimated that the peak load of the Achilles tendon during running is around 6 to 8 times the body weight (Kader et al., 2002). Therefore, an increase in the overload of the Achilles tendon due to a decrease in the activity of the muscles recruited for shock absorption could be interpreted as a negative adaptation for the runners with injured Achilles tendons. However, from the design of this study it cannot be concluded whether this result is an adaptive response to injury or an aetiological factor. Further prospective studies are needed to answer this question. There are few studies on injuries in the literature that have used a prospective design. A recent study that was designed to investigate a variety of injuries (Willems et al., 2007), showed that pronation excursion was associated with increased pressure on the medial side of the foot and this was associated with an increased risk of injury. However, the logistical difficulties conducting these types of large prospective studies are clear.

In another line of investigation, studies on running shoes with different hardness have found that muscle activity is adjusted according to the impact force to avoid muscle vibration (Wright et al., 1998; Nigg and Liu, 1999; Wakeling et al., 2002b; Boyer and Nigg, 2004). The natural frequency of the lower extremity soft tissues is between 5 and 65 Hz and the impact force frequency remains between 10 to 20 Hz (Wakeling et al., 2001; Nigg and Wakeling, 2001). The reduction in muscle activity in the injured compared to the uninjured group observed in this present study may indicate that the injured group can experience a larger muscle vibration (a resonance phenomena), as the muscle frequency would be more similar to the impact force frequency. It has been speculated that the increase in muscle activity would increase the resonance frequency and the damping coefficient in the soft tissues. Therefore, changes in lower extremity muscle activity would alter joint loading and this could be a potential modality for treatment and prevention of the injury (Nigg, 2001; Wakeling et al., 2002a). However, caution is necessary over the interpretation of the data. In this present study integrated EMG (IEMG) or the sums of muscle activity over a period of time were measured and not EMG frequency as in previous studies where this theory

was reported (Nigg, 2001; Wakeling et al., 2002a). Nevertheless, it is plausible to suggest from the results of this present study that rehabilitation programs that stimulate muscle activity may reduce the internal load on soft tissues.

In this study, it was found similar muscle activity of the LG and PE muscles between groups. This was not a predicted result as a higher activity of the plantar-flexor muscles was expected as a result of increased tendon stiffness in the injured group. However, because this study only measured activity in the lateral gastrocnemius muscle, it is therefore suggested that muscle activity in the soleus, as well as the medial gastrocnemius, be investigated in future studies.

In conclusion, the results from this case-control study shows that reduced muscle activity is an intrinsic factor that is associated with Achilles tendinopathy in runners. Additionally, considering that the impact forces were similar between the two groups and that muscle activity was lower in the injured group, it can be speculated that the injured group was experiencing lower shock absorption compared to the uninjured group. Therefore, it can be postulated that rehabilitative exercises designed to strengthen the muscles or other mechanisms (e.g. footwear) that increase muscle activity may be beneficial for runners with Achilles tendinopathy.

Although biomechanical (kinematic, kinetic and muscle activity) parameters are important factors to consider in Achilles tendinopathy, more recently the variability in these biomechanical parameters have been proposed as novel intrinsic risk factors that may be associated with running injuries (Hamill et al., 1999; Heiderscheit et al., 2002). This has, to our knowledge, never been studied in runners with Achilles tendinopathy. Therefore, variability in these biomechanical parameters as possible risk factors for Achilles tendinopathy will be investigated in the next study.

CHAPTER 6. STUDY 3: VARIABILITY IN BIOMECHANICAL (KINETIC, KINEMATIC AND MUSCLE ACTIVITY) PARAMATERS AS RISK FACTORS ASSOCIATED WITH ACHILLES TENDINOPATHY IN RUNNERS

6.1 Introduction

Variability of data, both within and between participants, is normally interpreted as "noise" and several techniques are used to reduce or eliminate this "noise" during data collection in the laboratory setting (Burden et al., 2003; Hunter et al., 2004; Schwartz et al., 2004). However, variability is inherent within and between any biological systems (Newell and Corcos, 1993). Studies to examine intra-participant variability in biological parameters have been developed in several disciplines including cardiology, sports science and biomechanics. In cardiology, it has been found that a decrease in heart rate variability is associated with cardiac pathologies (Huikuri et al., 1999). In the sports sciences it has been shown that overtrained athletes have a decrease in heart rate variability during standing (Uusitalo et al., 2000). Recently, this inherent variability has been studied in biomechanical system and may be a mechanism that is associated with injury risk (Hamill et al., 1999; Heiderscheit et al., 2002). Therefore, the possible association between variability in biomechanical parameters and risk for Achilles tendinopathy is the focus of this Chapter.

Within participant variability (intra-participant variability) for kinetic, kinematic and muscle activity parameters has been studied for many years during walking and running movements in uninjured populations (Bates et al., 1983; Kadaba et al., 1985; Ferber et al., 2002; Schache et al., 2002; Queen et al., 2006). However, there are only a few reports where intra-participant variability comparing uninjured and injured runners (Hamill et al., 1999; Heiderscheit et al., 2002; Ferber et al., 2005).

One of the early studies, which investigated intra-participant variability in uninjured runners, suggested that eight trials per participant were necessary to maintain constant participant condition values (Bates et al., 1983). Other studies found that sagittal plane kinematics parameters have a lower variability compared with frontal or

transverse plane kinematics parameters during walking and running movements (Kadaba et al., 1989; Queen et al., 2006). Some studies have also found that vertical and anterior-posterior forces are less variable than medio-lateral forces. EMG data have been shown to be less variable when measured within a day, but have greater variability when measured on consecutive days (Kadaba et al., 1989). Furthermore, EMG data are more variable if surface electrodes are used rather fine wire electrodes (Kadaba et al., 1985).

According to Schache et al. (2002), variability between trials in three dimensional movement analyses can be influenced by four main factors: 1) instrumental errors, which may be manifested with random variations; 2) the use of kinematic models that usually have associated assumptions; 3) skin movement artefacts; and 4) the inherent variability that is present in biological system, including any human movement.

In the area of variability in biomechanics of injury, it has been shown that uninjured runners have a higher kinematic variability compared to runners with patellofemoral pain (Hamill et al., 1999; Heiderscheit et al., 2002). The reason for the higher variability in uninjured runners is not clear, but it has been speculated that variability in kinematic parameters within subjects may assist on the attenuation of joint loading by a broader distribution of stress on different tissues (Hamill et al., 1999; Heiderscheit et al., 1999).

As mentioned previously, it has been postulated that sensory feedback on the sole of the foot is an important mechanism to sense force input signal and to promote movement adjustment in order to minimize soft tissue vibration (Nigg and Wakeling, 2001; Nurse and Nigg, 2001). Therefore, an increase in stride-to stride variability, that has been documented in uninjured runners (Hamill et al., 1999; Heiderscheit et al., 1999) may be associated with a better sense of the different force input signals that are sensed on the plantar surface, and/or a better transfer of this information to the central nervous system to promote a dynamic movement response. However, this assumption has to be treated with caution as not all the studies with injured populations had the same findings (Heiderscheit et al., 2002; Ferber et al., 2005).

Although intra-participant (stride to stride) variability in injured populations has been studied, the same cannot be stated for inter-participant (between participants) variability. According to our knowledge the possible association between inter-participant variability in biomechanical parameters and injury risk in runners has not been yet investigated. To date, inter-participant variability was mainly investigated when scientists were interested in validating biomechanical models (Griffin, 2001) or different methods of EMG normalization (Burden et al., 2003). Furthermore, some studies which have investigated shoes with different hardness have found a subject specific response in relation to kinetic, kinematics and energy cost parameters (Dufek et al., 1991; Nigg et al., 2003; Bishop et al., 2006; Kersting and Bruggemann, 2006b).

Therefore the aim of this study was to investigate whether intra- and inter-participant variability in kinetic, kinematic and EMG parameters is associated with Achilles tendinopathy in runners. Considering that intra-participant variability of biomechanical measurements is an important regulator which adjusts to internal and external variants, this study hypothesize that runners with Achilles tendon injuries would have less variability in biomechanical measurements between running strides (intra-participant variability) when compared with uninjured runners. It also hypothesises that between participants variability (inter-participant variability) would be lower in the Achilles injured population.

6.2 Methods

The methods related to this study have already been described in Chapter 3 – *Research Methodology*.

6.2.1 Data Analysis

Data were processed as described in section 3.3 of Chapter 3 *Research Methodology*. Two measurements of variability were used in this study: the intra-participant variability (or stride to stride variability); and inter-participant variability (or variability between participants).

Coefficient of variance (CV) was the method selected to measure intra- and inter-participant variability. This is a common method to measure variability and it represents the standard deviation as a percentage of the mean and it is valuable to compare variability of scores in different distributions. For this analysis, the coefficient of variation equation was adapted, as suggested by Burden *et al*. (2003), to calculate the variability of a continuous variable over the entire running cycle for the kinetics, kinematics and EMG parameters.

The equation of coefficient of variance is calculated as follows:

$$CV = \frac{\sqrt{\frac{1}{k}\sum_{i=1}^{k}\delta_i^{\,2}}}{\frac{1}{k}\sum_{i=1}^{k}\left|\overline{X_i}\right|}$$

where k is the number of time intervals over the gait cycle (i.e. 51 data points), $\overline{X_i}$ is the mean of the kinetic, kinematic or EMG values at the i^{th} interval of the gait cycle (each interval corresponded to 2% of the running cycle).

The main outcome parameters that were chosen in this component of the study were as follows: 1) kinetic variables - vertical ground reaction forces and anterior-posterior ground reaction forces; 2) kinematic variables - hip, knee and ankle sagittal plane kinematics; 3) Muscle activity - tibialis anterior, peroneus longus, lateral gastrocnemius, rectus femoris, biceps femoris, and gluteus medius. As previously stated, these variables were analysed over the entire gait cycle.

All the kinetic, kinematic and muscle activity parameters were calculated over five trials for the intra-participant variability in the injured and uninjured group. For the inter-participant variability the CV of an outcome parameter was calculated for the 34 uninjured participants and the 21 injured participants using the ensemble average of the participants from each group. To calculate the coefficient of variance for intra-

and inter-participant variability a set programme was created and processed in Matlab™ (Math Works, Natick, MA).

6.2.2 Statistical analysis

An independent t-test was applied to compare intra-participant variability (stride to stride) between the injured and uninjured groups. For the inter-participant variability, a single value was produced for each group (injured and uninjured), therefore no statistical test were performed. The differences between the groups for inter-participant variability are depicted as a percent difference (%).

6.3 Results

6.3.1. Intra-participant variability of kinetic, kinematic and muscle activity parameters

The intra-participant coefficient of variance for kinetic and kinematic parameters was similar between injured and the uninjured participants (Table 6.1).

Table 6.1. Intra-participant coefficient of variance (CV) of kinetic and kinematic parameters in the injured and uninjured groups (Values are depicted as mean ± SD).

Parameters	Uninjured (N=34)	Injured (N=21)	p value
Anterior-posterior forces	0.62 ± 0.13	0.57 ± 0.17	0.060
Vertical forces	0.24 ± 0.08	0.25 ± 0.10	0.737
Ankle sagittal plane kinematics	0.24 ± 0.09	0.33 ± 0.31	0.346
Knee sagittal plane kinematics	0.08 ± 0.02	0.16 ± 0.32	0.352
Hip sagittal plane kinematics	0.09 ± 0.05	0.13 ± 0.16	0.272

The intra-participant coefficient of variance for muscle activity parameters was similar between the injured and uninjured participants (Table 6.2).

Table 6.2. Intra-participant coefficient of variance (CV) of muscle activity in the lower limb muscles in the injured and uninjured groups (Values are depicted as mean ± SD).

	Uninjured (N=34)	Injured (N=21)	p value
Tibialis Anterior	0.39 ± 0.26	0.32 ± 0.09	0.129
Biceps Femoris	0.43 ± 0.21	0.41 ± 0.13	0.681
Peroneus Longus	0.45 ± 0.20	0.45 ± 0.17	0.929
Gluteus Medius	0.43 ± 0.14	0.44 ± 0.16	0.791
Lateral gastrocnemius	0.50 ± 0.29	0.45 ± 0.16	0.395
Rectus Femoris	0.44 ± 0.19	0.46 ± 0.16	0.700

6.3.2. Inter-participant variability of kinetic, kinematic and muscle activity parameters

A single value was produced for each parameter in each group. The results for inter-participant variability in the injured and uninjured groups are presented in Table 6.3 and 6.4. Inter-participant variability (injured vs. uninjured) was more than 25% higher in the uninjured than the injured group for the horizontal and vertical forces (Table 6.3). Kinematic parameters were pratically similar for the ankle and hip sagittal plane kinematics (Difference = -0.9 % and 9.3 % for ankle and hip sagittal plane kinematics, respectively). However, for the knee kinematics, there was an opposite relationship and coefficient of variance was higher for the injured group (Difference = -38.9 %).

Table 6.3. Inter-participant coefficient of variance (CV) of kinetic and kinematic parameters in the Injured and Uninjured groups (Values are depicted as mean and the % difference in variability between groups for each parameter is depicted).

	Uninjured (N=34)	Injured (N=21)	Difference (%)
Anterior-posterior forces	0.65	0.47	27.9
Vertical forces	0.29	0.20	30.1
Ankle sagittal plane kinematics	0.20	0.21	-0.9
Knee sagittal plane kinematics	0.08	0.11	-38.9
Hip sagittal plane kinematics	0.09	0.09	9.3

The coefficient of variance was higher for the uninjured group than the injured group for all the six muscles measured (Table 6.4). The range of difference of the coefficient of variance varied between 37.1% and 69% amongst the different muscles.

Table 6.4. Inter-participant coefficient of variance (CV) of muscle activity parameters in the Injured and Uninjured groups (Values are depicted as the mean and the % difference in variability between groups for each parameter is depicted).

	Uninjured (N=34)	Injured (N=21)	Difference (%)
Tibialis Anterior	0.68	0.21	68.5
Biceps Femoris	0.74	0.29	60.0
Peroneus Longus	0.56	0.35	37.1
Gluteus Medius	0.96	0.31	67.2
Lateral gastrocnemius	0.58	0.35	40.4
Rectus Femoris	1.01	0.31	69.0

6.4 Discussion

The two main findings of this study were that intra-participant variability (stride-to-stride variability) for kinetics, kinematics and muscle activity parameters was not associated with Achilles tendinopathy in runners (Tables 6.1 and 6.2) but inter-participant variability for kinetic variables and muscle activity parameters was lower in runners with Achilles tendinopathy. (Table 6.3 and 6.4).

Based in the results from previous studies, it was expected that intra-participant variability (stride-to-stride variability) would be higher in the uninjured than in the injured group (Hamill et al., 1999; Heiderscheit et al., 1999). The rationale behind this assumption was that a greater stride-to-stride variability would alternate the load on different tissues during each stride and hence, protect tissues from developing an overuse injury. In these studies, it was also suggested that the reduced variability in the injured runners could be associated with pain experienced during running. This concept is supported by results from one study (Heiderscheit, 2000) where it was observed that when individuals with unilateral patellofemoral pain were treated with patellar taping to reduce pain, an increase in variability was observed.

In this present study, variability between strides was not statistically different between the injured and the uninjured groups (Tables 6.1 and 6.2), rejecting the initial hypothesis and contradicting the findings from previous studies (Hamill et al., 1999; Heiderscheit, 2000; Heiderscheit et al., 2002). This finding may be related to a number of factors. Firstly, this study specifically chose to exclude more severely injured runners (Grades III and IV) so that they do not have pain when tested in the laboratory to measure biomechanical variables (with the exception of a specific prolonged running protocol which is described later where they were required to run until they developed pain – Chapter 8). Therefore, the grade of injury in this study (Grades I and II – pain only after running or during running but not restricting running) may not have been sufficiently severe to result in significant differences in stride to stride variability when compared with the uninjured population.

Secondly, it is important to note that the results of this study do support the findings from one other study where also no statistical differences in variability was found when runners with various lower extremity injuries, who were tested without orthotics or with inverted orthotics, were compared with control runners (Ferber et al., 2005). In that study, the failure to show a decreased variability in the injured runners was however explained by the fact that the injured group consisted of a variety of running injuries. It was suggested that this might increase the variability between participants and, therefore, reduce the power to detect differences. Furthermore, in that study, the researchers investigated a specific pattern of movement of the rearfoot-tibia coupling, which reflects the movement rearfoot eversion-inversion and tibia internal and external rotation, and this was a parameter that has not been investigated in previous studies.

Thirdly, it has also been suggested that intra-participant variability may be affected by differences in the peripheral sensory information (Ganevia and Gurke, 1992). It has been shown that the mechano-receptors on the sole of the foot are an important factor that may determine variability within the locomotor system (Nurse and Nigg, 2001). Intra-participant variability was significantly higher when runners ran barefoot, compared to running with two footwear conditions (Kurz and Stergiou, 2003). This finding suggests that the increase in sensory feedback in the barefoot condition results in increased intra-participant (stride to stride) variability (Kurz and Stergiou, 2003). The results of the present study showed that runners with longstanding Achilles tendinopathy have similar intra-participant (stride to stride) variability when compared to uninjured runners. Therefore, it is also possible that the injured runners in this study have similar mechanisms to alternate load in different tissues from stride to stride during running because they have had the injury for a long time and were able to adapt to changes in sensory input during running. However, as far as can be determined, there are no studies where the intra-participant variability of injured runners has been measured in conditions with altered sensory inputs. Therefore, further studies should investigate if alteration of the external environment (e.g. running shoes, running surface) or other sensory inputs (e.g. pain) would affect movement variability in injured and uninjured runners.

The novel finding of this study was that injured runners, as a group, appeared to exhibit lower inter-participant variability in selected kinetic and muscle activity parameters compared with uninjured runners (Tables 6.3 and 6.4). Therefore, reduced inter-participant variability in selected kinetic and muscle activity parameters during running are novel intrinsic factors that are associated with Achilles tendinopathy in runners. The importance of this finding is that similar responses to kinetics and muscle activity parameters that were observed in this study between the injured runners might indicate that a similar general mechanism predisposed them to injury.

In most reports, only intra-participant variability (or stride-to-stride) variability between injured and control runners has been studied (Hamill et al., 1999; Heiderscheit et al., 2002). As far as is known, the present study is the first study which examined inter-participant variability between an injured and an uninjured runner population. In the present study, inter-participant variability (uninjured group vs. injured group) was consistently (>30%) lower for kinetic parameters (anterior-posterior and vertical forces) in the injured group compared with the uninjured group. Similarly, for muscle activity parameters, inter-participant variability was consistently lower (> 37%) in the injured compared to the uninjured group. Therefore, with the exception of the knee and ankle sagittal plane kinematics, inter-participant variability for all the tested parameters was lower in the injured runners.

The finding that inter-participant variability in vertical and anterior-posterior impact forces were less in the injured runners may indicate that these runners tend to experience less variable forces during running, when compared to uninjured runners (Table 6.3). The reduction in inter-participant variability in the impact forces between the injured participants may be related to the finding of reduced inter-participant variability that was observed in muscle activity for all the muscles during the gait cycle (Table 6.4). If this result is interpreted using the impact force and muscle tuning paradigm (Nigg, 2001), it could be suggested that less variability in the force input, as sensed by the mechanoreceptors on the sole of the foot, may result in less variability in the information that is transmitted to the central nervous system, and this may result in less variability in lower limb muscle activity.

It has previously been stated that muscle activity of the tibialis anterior is adjusted according to external conditions, and that muscle activity is for example altered when wearing shoes or when wearing "unstable shoes" (Nigg, 2004), compared to running barefoot (von Tscharner et al., 2003). Therefore, it can be hypothesised that uninjured runners experienced more variability in impact forces, even though external conditions were similar between the two groups (shoe, surface and running speed). The variable impact forces then resulted in variability in the muscle activity of the lower limb muscles, in order to adjust to the range of impact forces.

It is also an interesting observation that inter-participant variability in joint kinematics was not similar in all joints. More similar variability between groups was observed for ankle and hip kinematics, whereas greater variability in kinematics of the knee joint was observed in the injured group. This result is difficult to explain and would require further study.

It has been documented that there is a high inter-participant variability in biomechanical and energy cost parameters in injured runners who run with shoes of different hardness (Dufek et al., 1991; Nigg et al., 2003; Bishop et al., 2006; Kersting and Bruggemann, 2006b). It has been suggested that this high inter-participant variability may be associated with other factors such as differences in lower limb and foot morphology, functional behaviour or differences in the sensitivity to external signals between participants in the sensitivity to external signals (Nigg et al., 1998). Therefore, it is important to consider these as possible explanations for the observations of increased inter-participant variability in the uninjured runners. Firstly, injured runners could exhibit reduced lower inter-participant variability of the lower limb and/or foot morphology and this might be related to the lower inter-participant variability observed in this study for the kinetic and muscle activity parameters. The coefficients of variance of all the anthropometrical parameters that were measured (Chapter 4. Study 1: *Training, Flexibility and Lower Limb Alignment Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*), were therefore calculated and are presented in Appendix 12. These results of this analysis show that the difference in the coefficients of variance is quite variable between injured and uninjured runners, with no specific pattern. Therefore, it is unlikely that the reduced

inter-participant variability between the two groups is due to variability in lower limb morphology parameters.

Secondly, it is possible that variation in functional behaviour could be related to the reduced inter-participant participant variability in the injured runners that was observed. It is difficult to measure functional behaviour in runners and it may be speculated that measures of muscle strength, flexibility or joint range of motion may define aspects of functional behaviour of a runner. Therefore, the variability in these parameters which were measure (Chapter 4. Study 1: *Training, Flexibility and Lower Limb Alignment Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*) was also analysed (Appendix 12). Inter-participant variability in subtalar range of motion, knee range of motion, and flexibility (sit and reach test) was consistently higher in the injured compared with the uninjured group. This observation may partially explain the increased inter-participant variability in knee and ankle kinematics that was observed in the injured runners. However, it does not explain the increased inter-participant variability that was observed in the kinetic and muscle activity parameters in the uninjured runners.

Finally, it is possible that injured runners have a reduction in the sensitivity to external or maybe even internal sensory signals, and this could explain the reduction in inter-participant variability that was observed in this study. Two sensory signals during running that are important to consider are: 1) the effect of shoe midsole hardness; and 2) the development of pain during running. In the final two experimental chapters of this thesis the effect of altering these two parameters on lower limb biomechanics (kinetics, kinematics and muscle activity) will be investigated (Chapters 7 and 8). However, the analysis of how these sensory inputs may affect variability is beyond the scope of his thesis and this will have to be investigated in future.

This study showed that reduced inter-participant, but not intra-participant, variability in selected kinetic, kinematic and muscle activity parameters in injured runners during running are novel intrinsic factors associated with Achilles tendinopathy in runners. The possible reasons for this observed reduction in inter-participant variability in injured runners is not known and would require further investigation. One possible

explanation for this observation is that there may be differences in the sensitivity and responses of injured runners to sensory input. However, one possible limitation of this study was that the sample size was smaller in the injured compared to the uninjured group. This may have resulted in a smaller variation between participants. Therefore, further studies should analyse inter-participant variability using a similar sample size between groups.

The results of this study are novel, and therefore raise many questions and a number of areas for further study can be identified. Further analysis on functional behaviour, including measures of muscle strength and a more comprehensive flexibility test may be necessary. Furthermore, further examination on variability in response to altered external (e.g. shoes) or internal sensory signals (e.g. pain) should be conducted. In addition, further prospective studies should be conducted to determine if individuals with low movement variability indeed have a higher risk of injuries compared with those that have a high variability. There is also a need to study whether movement variability may be affected during the rehabilitation phase and after healing. Finally, there is a need to study the movement variability of injuries in sports other than running only to establish a stronger link between movement variability and sports injury.

CHAPTER 7. STUDY 4: THE EFFECT OF DIFFERENT SHOE HARDNESS ON LOWER LIMB KINETIC, KINEMATIC AND MUCSLE ACTIVITY VARIABLES IN RUNNERS WITH AND WITHOUT ACHILLES TENDINOPATHY

7.1 Introduction

Running shoes have been indicated as one of the extrinsic factors related to injury (Frederick, 1986; Barnes and Smith, 1994; Lake, 2000; Johnston et al., 2003). The association between running shoes and running injuries has been linked to a number of factors including the reduction of impact force (Dickinson et al., 1985; De et al., 1994; De Wit et al., 2000), control of pronation (Clarke et al., 1983b; Nigg and Segesser, 1986; Nigg and Morlock, 1987; Nigg et al., 1987) and more recently to their possible effect on muscle activity (Nigg and Liu, 1999; Wakeling et al., 2001; Wakeling et al., 2002b; Nigg et al., 2003; von Tscharner et al., 2003; Nigg et al., 2006a). However, in a systematic analysis (Yeung and Yeung, 2001b), it has been shown that the precise relationship between running shoes or insoles and the prevention of running injuries is still uncertain and requires further investigation.

Shoes have different proprieties that may affect biomechanics. Midsole hardness however, is the one shoe characteristic that is most widely investigated (Clarke et al., 1983a; Snel et al., 1985; Nigg et al., 1987; Milani et al., 1997; Lake and Lafortune, 1998; Hardin et al., 2004). It is suggested that shoes with a harder midsole will result in a higher vertical impact force during running. Impact tests performed by dropping weights or pendulums on shoes have shown that impact forces are reduced in softer compared with harder shoes (Aerts and De, 1993). However, there is a poor correlation between these results from material mechanical tests and running kinetics when tests are conducted in runners (Clarke et al., 1983a; Snel et al., 1985; Kalin et al., 1988a). It has been shown that peak vertical impact forces are not affected by shoes (Clarke et al., 1983a; Nigg et al., 1987; Komi et al., 1987; Wright et al., 1998; Hardin et al., 2004) or surfaces (Dixon et al., 2000; Kerdok et al., 2002; Hardin et al., 2004) with different hardness. However, it has been shown that vertical loading rate can be affected by shoes (Clarke et al., 1983a; Wright et al., 1998). The similarities in

vertical impact forces despite different running shoe conditions are currently explained by the idea that kinematic or muscle activity adjustments are observed during running (Bobbert et al., 1992; Ferris et al., 1999; Hardin et al., 2004; Boyer and Nigg, 2004).

It has been reported that an increase in shoe hardness would increase the rate of knee flexion (Wright et al., 1998), peak ankle dorsiflexion velocity (Hardin et al., 2004) and maximum rate of foot pronation (Luethi et al., 1987). Furthermore, it has been shown that EMG activity before, or after, heel strike is altered when running with shoes with different hardness (Wakeling et al., 2002b; Nigg et al., 2003; von Tscharner et al., 2003). Nevertheless, there is no evidence that biomechanical adjustments, in particular muscle activity responses occur with different shoe hardness in an injured population.

In previous chapters (Chapters 4 and 5) intrinsic factors that are associated with Achilles tendinopathy (e.g. self-reported previous injury, running for more years, reduced muscle activity of tibialis anterior and rectus femoris) were identified. Furthermore, in the previous chapter (Chapter 6) reduced inter-participant variability in kinetic and muscle activity parameters were also identified as a possible novel risk factor that is associated with Achilles tendinopathy in runners.

It has been suggested that this observation may be related to differences in the sensitivity and responses to external sensory input such as by altering footwear. Therefore, the aim of this study was to investigate the effect of differences in shoe hardness on biomechanical parameters (including kinetics, kinematics and changes in muscle activity responses) in runners with Achilles tendinopathy. It was hypothesized that shoes with different midsole hardness will alter lower limb kinetic, kinematic and muscle activity responses in runners with Achilles Tendinopathy.

7.2 Methods

The methods used in this particular investigation were described in Chapter 3 - *Research Methodology*. The main methodological difference in this study was that the

participants performed the running trials in two different shoe conditions: 1. Soft midsole shoe: Shore A = 40 (Figure 7.1); 2. Hard midsole shoe: Shore A = 50 (Figure 7.2) (sizes 6 to 12, Rainha, Alpargatas Inc, Brazil). The hardness of the midsole was measured with a durometer that was applied to the midsole (Figure 7.3).

Figure 7.1. : Illustration soft shoe

Figure7.2: Illustration hard shoe

Figure 7.3: Illustration of a durometer.

The order of the shoe conditions was randomly assigned and the participants performed 10 running trials at a self-selected speed wearing each running shoe with no socks or orthotics. The period between each treatment was short (time to change the shoes, attach the reflective markers on the shoe and reprocess the subject calibration). Data analysis was processed according to the methods explained in section 3.3, Chapter 3 – *Research Methodology*.

7.2.1 Statistical Analysis

All data are expressed as mean ± standard deviation ($X \pm s$). Differences between the two groups and running shoe conditions were compared using Repeated Measure ANOVA. Significant main effects were analysed further with a Tukey *post hoc* analysis test. The false discovery procedure was used to account for multiple comparisons (Curran-Everett, 2000). Statistical significance was accepted as $p \leq 0.05$ before the adjustments were made. The level of significance displayed in the tables represents the correct level (i.e. after the adjustment for multiple comparisons). To measure the magnitude of a treatment effect, the effect size was reported when statistical differences were observed. The Eta squared (r^2) was the method selected to measure the effect size whereby small effect size, $r^2 = 0.010$; medius, $r^2 = 0.059$ and large, $r^2 = 0.138$ (Tabachnick and Fidell, 1989).

7.3 Results

There were no significant differences between the type of shoes and groups (uninjured vs. injured) for the temporal distance parameters (Table 7.1) and vertical and anterior-posterior forces (Table 7.2). Kinematic variables were also not significantly different between shoes or groups (Table 7.3).

Table 7.1. Temporal distance variables in injured and uninjured runners in the soft and hard running shoe condition (Values are depicted as mean ± SD).

	SOFT SHOE		HARD SHOE		
	Uninjured (N=34)	**Injured (N=21)**	**Uninjured (N=34)**	**Injured (N=21)**	**p value**
Speed (m/s)	3.01 ± 0.30	2.97 ± 0.37	2.98 ± 0.37	2.99 ± 0.38	0.990
Stride Ln (m)	2.23 ± 0.28	2.17 ± 0.30	2.18 ± 0.27	2.09 ± 0.51	0.423
Stride F (strides/min)	82 ± 5	82 ± 7	83 ± 4	83 ± 6	0.940
CT (s)	0.17 ± 0.01	0.17 ± 0.02	0.17 ± 0.01	0.17 ± 0.01	0.941
VSDsp (cm)	9.80 ± 1.5	9.7 ± 2.1	9.7 ± 1.2	9.6 ± 1.9	0.982

Abbreviations: *CT = contact time; VSDsp = vertical sacral displacement*

Table 7.2. Kinetic variables in injured and uninjured runners in the soft and hard running shoe condition (Values are depicted as mean ± SD).

	SOFT SHOE		HARD SHOE		
	Uninjured (N=34)	**Injured (N=21)**	**Uninjured (N=34)**	**Injured (N=21)**	**p value**
HBF (BW)	0.22 ± 0.05	0.20 ± 0.05	0.20 ± 0.04	0.18 ± 0.04	0.030
HPF (BW)	0.16 ± 0.04	0.16 ± 0.04	0.16 ± 0.05	0.16 ± 0.03	0.850
VIF (BW)	1.33 ± 0.25	1.45 ± 0.23	1.34 ± 0.25	1.32 ± 0.23	0.282
VLR (BW/s)	42.76 ± 9.01	44.79 ± 11.27	40.77 ± 7.44	41.29 ± 9.61	0.339
VPF (BW)	2.22 ± 0.16	2.18 ± 0.23	2.22 ± 0.18	2.17 ± 0.22	0.323

Abbreviations: *HBF = horizontal braking force; HPF = horizontal propulsive force; VIF = vertical impact force; VLR = vertical loading rate; VPF = vertical propulsive force.*

Table 7.3. Kinematics variables in injured and uninjured runners in the soft and hard running shoe condition (Values are depicted as mean ± SD).

	SOFT SHOE		HARD SHOE		
	Uninjured (N=34)	**Injured (N=21)**	**Uninjured (N=34)**	**Injured (N=21)**	**p value**
Hic (°)	39.8 ± 9.5	42.4 ± 7.1	39.7 ± 10.5	42.7 ± 7.6	0.817
Hto (°)	2.1 ± 11.8	3.8 ± 5.5	0.7 ± 8.3	4.9 ± 7.0	0.611
HROM (°)	36.7 ± 11.6	38.7 ± 7.1	38.9 ± 6.7	37.8 ± 6.2	0.814
KswE (°)	15.8 ± 7.8	20.1 ± 7.7	16.7 ± 7.5	19.0 ± 7.8	0.617
Kic (°)	15.9 ± 7.4	20.2 ± 7.2	17.0 ± 7.7	19.0 ± 7.3	0.371
Kst (°)	41.4 ± 8.5	42.2 ± 4.8	42.0 ± 8.4	41.0 ± 6.7	0.942
KROM (°)	25.5 ± 4.5	22.0 ± 5.5	26.2 ± 5.08	21.9 ± 5.8	0.171
Asw (°)	-12.6 ± 4.8	-11.5 ± 5.6	-12.1 ± 4.3	-11.2 ± 5.0	0.545
Aic (°)	-11.0 ± 8.6	-11.5 ± 5.8	-12.2 ± 4.5	-11.1 ± 5.1	0.899
Ast (°)	-21.4 ± 9.2	-20.9 ± 3.5	-21.2 ± 4.5	-20.2 ± 3.0	0.986

Abbreviations: *Hic = hip angle at initial contact; Hto = hip angle at toe off; HROM = hip range of motion; KswE = knee angle at terminal swing phase; Kic = knee angle at initial contact; Kst = knee angle at stance phase; KROM = knee range of motion; Asw = ankle angle at swing phase; Aic = ankle angle at initial contact; Ast = ankle angle at stance phase*

Although not significant, following the correction for multiple comparisons, the pre-IEMG activity of tibialis anterior (TA pre) tended to be higher in the uninjured group with the soft running shoes compared with the injured group with the hard running shoe (p=0.016) (Table 7.4 and Figure 7.1).

Post-IEMG of the rectus femoris (RF post) was significantly lower in the injured group compared with the uninjured group in both shoe conditions ($p < 0.001$) (Table 7.4 and Figure 7.2). The reduced IEMG activity of RF in the injured group with the soft shoe was maintained during the stance phase (RF SP) when compared with the uninjured group in both shoe conditions (p=0.004) (Table 7.5). Associated with that, there was a lower peak EMG activity of RF (RF PSP) in the injured runners during

soft and hard shoe conditions compared with the uninjured runners in both shoe conditions ($p < 0.001$) (Table 7.6, Figure 7.2).

Table 7.4. IEMG activity (%*s) Pre (100 ms before initial contact), Post (100 ms after initial contact) in injured and uninjured runners in the soft and hard shoes conditions (Values are depicted as mean ± SD).

	SOFT SHOE		HARD SHOE		
	Uninjured (N=34)	**Injured (N=21)**	**Uninjured (N=34)**	**Injured (N=21)**	**p value**
TApre	21.0 ± 5.9	17.3 ± 5.9	20.8 ± 6.7	16.8 ± 5.2	0.016*
TApost	13.0 ± 7.2	12.2 ± 8.0	13.8 ± 7.7	13.2 ± 6.2	0.544
BFpre	17.8 ± 4.7	16.1 ± 7.9	17.9 ± 5.5	16.1 ± 7.9	0.736
BFpost	17.5 ± 7.6	18.5 ± 7.1	17.1 ± 7.0	17.5 ± 6.7	0.829
PEpre	11.3 ± 5.3	10.2 ± 4.5	10.1 ± 4.0	10.0 ± 4.3	0.748
PEpost	28.4 ± 8.4	25.3 ± 10.2	27.6 ± 8.4	23.6 ± 10.2	0.623
GMpre	12.3 ± 3.1	13.0 ± 5.9	11.8 ± 3.3	13.3 ± 5.9	0.576
GMpost	24.3 ± 10.2	18.1 ± 7.9	24.4 ± 10.9	17.0 ± 6.4	0.250
LGpre	9.6 ± 4.1	9.6 ± 5.4	11.2 ± 4.4	10.2 ± 5.6	0.658
LGpost	27.5 ± 8.2	23.7 ± 10.4	27.9 ± 8.1	22.0 ± 10.4	0.709
RFpre	13.1 ± 4.3	14.8 ± 6.5	12.4 ± 5.0	14.6 ± 5.8	0.030
RFpost	33.5 ± 8.3	21.6 ± 9.6	33.7 ± 8.3	21.2 ± 9.2	0.000*

* Tukey *pos hoc* analysis test indicate significant difference between soft uninjured and hard and soft injured; and significant difference between hard uninjured and hard and soft injured, after adjustment with the false recovery procedure.

Effect size RFpost $r^2 = 0.326$ (i.e. large effect size)

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

Table 7.5. IEMG activity of the whole gait cycle (GC) and in the stance phase (SP) (%*s) in injured and uninjured runners in the soft and hard shoe conditions (Values are depicted as mean ± SD).

	SOFT SHOE		HARD SHOE		
	Uninjured (N=34)	**Injured (N=21)**	**Uninjured (N=34)**	**Injured (N=21)**	**p value**
TA GC	12804 ± 401	12630 ± 777	12747 ± 476	12590 ± 822	0.455
TA SP	4272 ± 1571	4216 ± 1797	4463 ± 1460	4796 ± 2220	0.575
BF GC	12901 ± 659	12810 ± 625	12770 ± 707	12727 ± 607	0.769
BF SP	5942 ± 1179	6733 ± 1640	5999 ± 1170	6703 ± 2143	0.107
PE GC	13004 ± 490	12957 ± 598	12927 ± 571	12912 ± 552	0.993
PE SP	8682 ± 1745	8077 ± 2447	8587 ± 1522	8391 ± 2424	0.824
GM GC	12578 ± 492	12504 ± 648	12460 ± 414	12367 ± 603	0.798
GM SP	6700 ± 1649	6172 ± 1271	6545 ± 1434	6140 ± 2038	0.950
LG GC	12949 ± 533	12707 ± 584	12927 ± 494	12719 ± 551	0.301
LG SP	8996 ± 1898	8141 ± 2579	8979 ± 1809	8072 ± 2675	0.9257
RF GC	12643 ± 441	12639 ± 566	12665 ± 512	12630 ± 511	0.781
RF SP	8207 ± 1304	6183 ± 2010	8290 ±1516	6767 ± 2520	0.004*

* Tukey *pos hoc* analysis test indicate significant difference between soft injured and hard and soft uninjured, after adjustment with the false recovery procedure.

Effect size RF SP $r^2 = 0.201$ p (i.e. large effect size)

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

Table 7.6. Peak EMG activity in the stance phase (P SP) (%) and time of occurrence of peak EMG activity in the stance phase (T SP) (% gait cycle) in injured and uninjured runners in the soft and hard shoe conditions (Values are depicted as mean ± SD).

	SOFT SHOE		HARD SHOE		
	Uninjured (N=34)	**Injured (N=21)**	**Uninjured (N=34)**	**Injured (N=21)**	**p value**
TA PSP (%)	244 ± 81	211 ± 83	250 ± 98	204 ± 65	0.358
TA TSP (s)	52 ± 10	54 ± 1	52 ± 10	56 ± 15	0.616
BF PSP (%)	272 ± 82	314 ± 91	252 ± 79	285 ± 94	0.034
BF TSP (s)	63 ± 7	63 ± 7	61 ± 9	62 ± 11	0.915
PE PSP (%)	402 ± 93	381 ± 140	392 ± 100	378 ± 133	0.829
PE TSP (s)	61 ± 6	61 ± 9	62 ± 6	62 ± 9	0.877
GM PSP (%)	318 ± 126	269 ± 82	292 ± 123	244 ± 90	0.252
GM TSP (s)	59 ± 6	62 ± 8	62 ± 9	63 ± 13	0.627
LG PSP (%)	382 ± 115	366 ± 140	373 ± 107	347 ± 118	0.892
LG TSP (s)	64 ± 6	63 ± 9	63 ± 5	62 ± 8	0.867
RF PSP (%)	413 ± 94	305 ± 84	421 ± 119	282 ± 88	0.000*
RF TSP (s)	59 ± 5	54 ± 8	57 ± 4	57 ± 7	0.034

* Tukey *pos hoc* analysis test indicate significant difference between soft uninjured and hard and soft injured; and significant difference between hard uninjured and hard and soft injured, after adjustment with the false recovery procedure.

Effect size RF PSP $r^2 = 0.270$ (i.e large effect size)

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

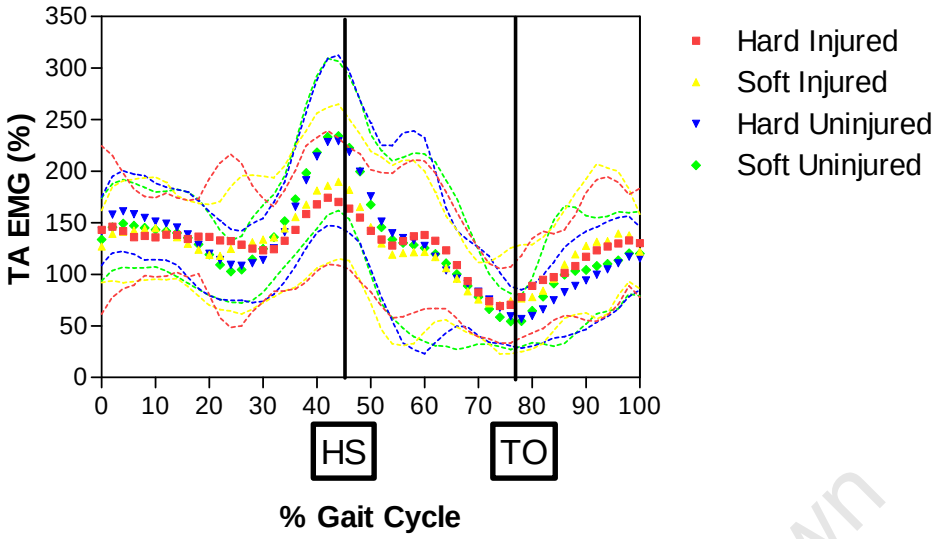

Figure 7.4. EMG of tibialis anterior (EMG TA) (%) of the injured and uninjured runners in the soft and hard shoe conditions. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

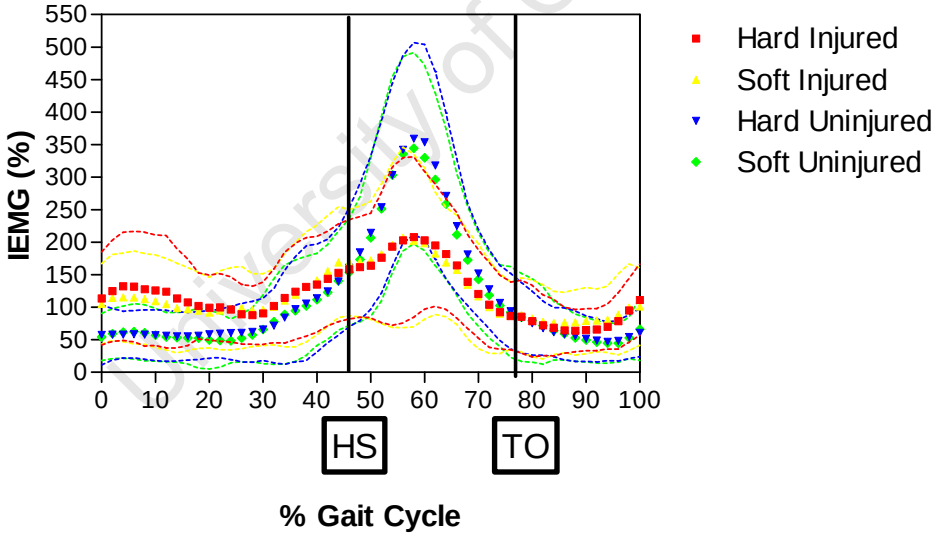

Figure 7.5. EMG of rectus femoris (EMG RF) (%) of the injured and uninjured runners in the soft and hard shoe conditions. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

7.4 Discussion

The main finding of this study was that muscle activity of the rectus femoris during the stance phase of running was consistently lower in the injured compared with the uninjured population, and that this effect was independent of the shoe condition (hard vs. soft). It was hypothesized that different shoe hardness could result in different biomechanical adaptations in injured and uninjured runners. However, the results of this study do not support this hypothesis as there were no significant differences in the kinetic and kinematic responses during the running cycle when using different shoes. Similarly, there was no significant differences EMG activity of lower limb muscles between the injured and uninjured groups when running with either hard or soft shoes.

In this study, similar impact forcers were observed when running in hard vs. soft shoes (Table 7.2). This findings is supported by results form previous studies, when uninjured runners ran with shoes of differing hardness (Clarke et al., 1983a; Snel et al., 1985; Komi et al., 1987; Wright et al., 1998). This observation of similar impact forces despite running with shoes of different hardness has always been attributed to kinematic adaptations that occur during foot contact (Luethi et al., 1987; Wright et al., 1998; De Wit et al., 2000; Hardin et al., 2004), however, in the present study, temporal distance and kinematic parameters were the same across different shoes (Table 7.1 and 7.3).

Although impact forces have been shown to be similar between different running shoe condition, some studies found a higher vertical impact forces can be different between an injured and uninjured population (Messier et al., 1991; Milner et al., 2006). In an early case control study (Messier et al., 1991), it was shown that injured runners (with patellofemoral pain) have a greater vertical propulsive force even when running at a slower running speed than an uninjured control group. Likewise, instantaneous and average vertical loading rate were significantly higher in female runners with a history of tibia stress fracture (Milner et al., 2006). However, in the present study there were no statistical differences in impact forces between runners with Achilles tendinopathy and uninjured runners. These findings are comparable to those findings already reported and discussed in Chapter 5. Study 2 *Lower Limb Kinetic, Kinematic and*

Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners, when runners were studied using a standard running shoe condition and to another case-control study with Achilles injured runners (McCrory et al., 1999). Furthermore, lower limb kinematics were expected to be different between runners with Achilles tedinopathy and uninjured runners (McCrory et al., 1999), but supporting the results from Chapter 5. Study 2 running kinematics were similar between the two groups of runners.

It was also hypothesized that shoes with different hardness would alter lower limb muscle activity responses differently in injured and uninjured runners. Previous studies have found altered muscle activity with different shoe hardness conditions in an uninjured population (Wright et al., 1998; Wakeling et al., 2002b; Nigg et al., 2003). In this study muscle activity was not altered according to different shoe conditions but it was constantly lower for rectus femoris in the injured group compared to the uninjured group (Tables 7.4 and 7.5 and Figure 7.5). IEMG of RF was significantly lower after heel strike in the injured group, when data were analysed 100 ms after heel strike or during the whole stance phase, independent of shoe condition. Moreover, peak EMG activity was significantly lower and occurred earlier in the injured group in the soft shoe condition than the injured group in both shoe conditions (Table 7.6).

The precise reasons for the observed reduction in EMG activity of RF during the stance phase and Achilles tendinopathy are not clear but deserve discussion. Firstly, it is known that RF acts eccentrically during the early stance phase to restrain the movement of the tibia (Novacheck, 1998b). It is therefore possible that increased Achilles tendon stiffness, perhaps due to injury may restrain the movement of the tibia, thereby reducing the muscle activity of the rectus femoris.

Secondly, muscle weakness, particularly the weakness of the gastrosoleus (Alfredson et al., 1998b; McCrory et al., 1999; Paavola et al., 2002) has been suggested as a risk factor for Achilles tendinopathy. Although muscle strength has not been measured in this present study, the reduction in IEMG activity of rectus femoris might indicate that this group may have a weakness in the quadriceps muscle. However, this result has to be interpreted with caution as although there is a linear relationship between

integrated EMG and muscle force during isometric contraction, this relationship is non-linear for concentric or eccentric contractions (Hof, 1997).

Thirdly, it is possible that the reduction in muscle activity could be in response to altered sensory feedback from afferents in the lower limb. Previous studies have shown that sensory feedback on the sole of the feet can alter muscle activity and plantar pressure distribution (Nurse and Nigg, 2001). Although it was expected that the different shoes hardness would possibly alter sensory feedback on the sole of the foot and thereby change muscle activity in injured and uninjured runners, this was not observed. Rather, in participants with Achilles tendinopathy, muscle activity was reduced in both shoe conditions. It is possible that the injury itself could have resulted in altered sensory activity, including the development of pain, which then caused a reduction in muscle activity independent of the variation of external sensory input (different shoe hardness). This however requires further investigation.

There was also a trend (significant before correction) to increase IEMG activity of tibialis anterior (TA Pre) in the uninjured group with the soft running shoes compared with the injured group with the hard running shoe (Table 7.4 and Figure 7.1). Previous study have shown that IEMG activity of TA just before heel strike was significantly higher when participants wore shoes compared to a barefoot condition (Von, V et al., 2003). Similary, the results of the present study showed that more cushioning (soft shoe) tended (not statistically significant) to increase IEMG activity of TA. Although caution have to be taken on the interpretation of this finding as the differences in TA pre IEMG activity occurred between runners of different groups (injured vs. uninjured).

It has to be considered that kinetic and kinematic parameters may vary non-systematically with midsole hardness (Kersting and Bruggemann, 2006a), perhaps indicating that different strategies were used by individuals to adapt to different shoe conditions. Additionally, it is important to note that IEMG responses to different shoe hardness is subject specific (Nigg et al., 2003). The variability in biomechanics response between individuals might have affected the results and were discussed in Chapter 6. Study 3 *Variability in Biomechanical (kinetic, kinematic and muscle*

activity) Parameters as Risk Factors Associated with Achilles Tendinopathy in Runners.

Finally, one limitation of this study should be considered. The difference in the midsole hardness between the two shoes that were used (10 shore A, difference), was perhaps not large enough to alter the variables that was studied. This could account for the failure to show differences between the two shoe types. In previous studies (Wakeling et al., 2002b; Nigg et al., 2003; Hardin et al., 2004), running biomechanical variables were studied in shoes with larger differences in midsole hardness (shore differences of 20 to 30 shore A). Furthermore, as has been previously stated, small differences in midsole density may not be perceived by individuals (Lake and Lafortune, 1998). Therefore, in this study the relatively small differences in midsole hardness may not have been perceived by the runners, thereby reducing possible differences in sensory feedback on the sole of the foot. It is therefore suggested that further studies should use shoes with a greater difference in their midsole hardness.

Based on the results of this study, it can be concluded that regardless of shoe hardness, within a narrow range, the injured runners had similar impact forces and kinematics but reduced muscle activity of RF during running. This suggests that injured runners might have less protection against impact load, and that shoes with more shock absorbing properties do not necessarily alter muscle activity in injured runners. This has practical implications in prescribing footwear in the management of runners with Achilles tendinopathy as increasing the cushioning of the shoes in the range presented in this study (Shore A between 40 and 50) does not to affect the biomechanics of these runners. Further investigations to determine the effect of altered sensory input, including pain, on muscle activity during running are required and this will be investigated in the next chapter.

CHAPTER 8. STUDY 5: THE EFFECT OF PAIN DEVELOPMENT DURING RUNNING ON LOWER LIMB KINETIC, KINEMATIC AND MUSCLE ACTIVITY VARIABLES IN RUNNERS WITH ACHILLES TENDINOPATHY

8.1 Introduction

The classic symptoms of Achilles tendinopathy are early morning stiffness and pain which progresses as loading increases. Other clinical features are a decrease in range of motion, swelling and weakness during activity (Scioli, 1994). The cause of pain in Achilles Tendinopathy is still unknown (Khan et al., 2000). An in depth discussion of the causes of pain in runners suffering from Achilles tendinopathy is beyond the scope of this thesis. However, it is important to briefly understand the mechanisms that may cause pain in Achilles tendinopathy. The reason for this is that pain development during running may alter sensory input and subsequently change motor (muscle activity) responses during running. These responses may be responsible for alterations in running biomechanics.

In the past, the pain associated with Achilles tendinopathy has been attributed to inflammation of the tendon (tendinitis). However, over the past decade it has been shown that there is no evidence of inflammatory cells and that the prostaglandin concentrations are not higher in patients who suffer from chronic tendon pain (Alfredson et al., 1999). It has also been suggested that the pain in Achilles tendinopathy may result from the separation of collagen fibers, but studies have found no association between collagen damage and pain (Alfredson and Lorentzon, 2000). Tissue degeneration has also been considered one of the possible mechanisms of pain, but it is well documented that degenerated tendons can be asymptomatic (Fredberg and Bolvig, 2002). More recently, it has been speculated that noxious components (e.g. glutamate) (Alfredson et al., 1999) or neovascularisation of the tendon (Alfredson et al., 2003) may stimulate the pain receptors of the tendon, and this causes the pain.

It is commonly accepted that runners with Achilles tendinopathy develop progressive pain during running. It is important to consider that this may affect lower limb biomechanics and muscle activity. However, to our knowledge this has not been systematically studied.

In one study in runners with Achilles tendinopathy, the subjects underwent a 5 min run on a treadmill. In this study, maximum pronation, time to maximum pronation and maximum pronation velocity were significantly higher in the Achilles tendinopathy group compared with a control group (McCrory et al., 1999). However, in this study it was not documented whether runners experienced any pain during running. In another study, electromyographic (EMG) activity of the quadriceps muscle was measured in female runners with and without patellofemoral pain syndrome during treadmill running (MacIntyre and Robertson, 1992). In this study, the differences between injured and non-injured runners were not higher than two standards deviations, and were not considered statistically significant. However, the researchers in both these studies did not document the pain responses in the injured group during running and could therefore not determine if the observed changes were as a result of pain development during running.

Thus, to our knowledge, there are no data from studies to indicate whether lower limb kinematic, kinetic or muscle activity parameters change as pain develops during running. Therefore, the aim of this study was investigate whether the development of pain during running is associated with changes in lower limb kinematics and muscle activity in runners with Achilles tendinopathy. It is hypothesized that the development of pain will alter lower limb biomechanics and muscle activity in runners with Achilles tendinopathy when compared to responses in uninjured runners.

8.2 Methods

Participants' characteristics involved in this study have already been described in detail Chapter 3 – *Research Methodology*. The general experimental procedures were also described in the same chapter. However, because participants in this study ran on a treadmill ground reaction force data were not collected.

8.2.1 Running Trial

The running trials were conducted on a treadmill (Life Fitness T3-0, Illinois, United States) with standardized running shoes (Rainha Athens, Alpargatas Inc; shore A 40, sizes 6 to 12) and the participants did not use socks or orthotics.

After a familiarization run for 2 or 3 mins, the treadmill was adjusted to a self-selected running speed. The participants were required to perform the trial for at least 10 mins or until the first symptom of injury discomfort appears (injured group). Kinematic and EMG data were recorded at 30s, 3 mins, 6 mins, 9 mins of the running trial for the uninjured group and until pain was felt in the injured group. Because runners in the injured group had variable pain responses, the numbers of recorded trials varied in the Achilles tendinopathy group. The number of recorded trials at the time interval for the injured group is listed in Table 8.1, and the time when pain condition occurred is listed in Table 8.2. Each trial was recorded for 10 seconds.

Table 8.1. Number of trials injured group.

Time interval	30 s	3 min	6 min	9 min	Pain
Number of trials	21	18	17	9	21

Table 8.2. Number of participants that felt pain in each time range

Time range	1 to 3 min	4 to 6 min	7 to 9 min	10 to 15 min
Number of participants	3	1	8	9

8.2.2 Data Analysis

Data were analyzed for the right limb of the uninjured runners and for the injured limb in the injured runners. As one of the outcome measurements was the EMG at 100 ms before and after heel strike, the data were processed for one stride length of the opposing limb. Data for uninjured runners and runners injured on the right limb

were processed from left heel strike to left heel strike. For runners who were injured on the left limb, data were processed from right heel strike to right heel strike.

Kinematic data processing was similar to that already described in Chapter 3- *Research Methodology*. However, some temporal distance parameters were processed differently. Step length was determined by the horizontal displacement of the heel marker of the opposing leg (Figure 8.1). To determine the stride length, this result was multiplied by two.

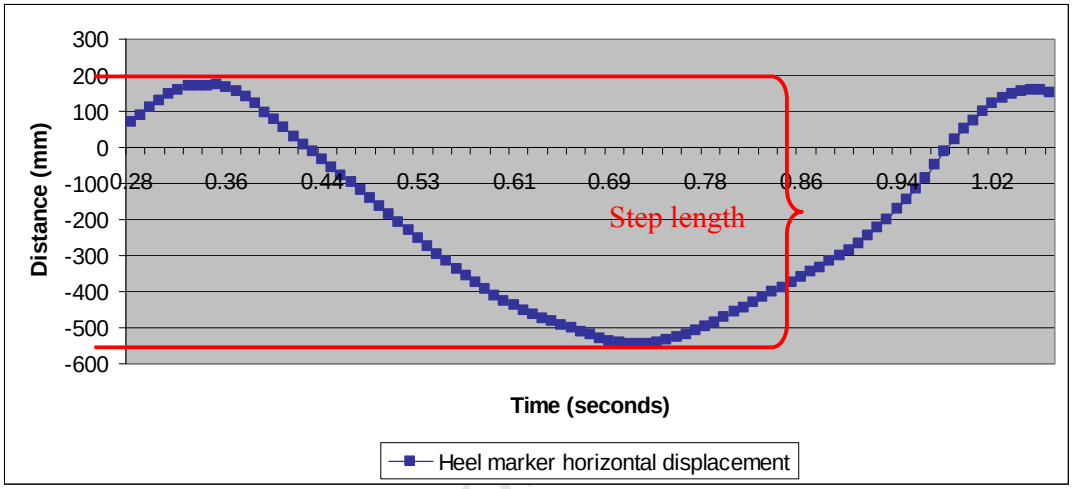

Figure 8.1. Typical horizontal displacement-time graph of the heel markers of a treadmill trial.

Heel strike and toe off of the leg of interest were determined by the vertical displacement of the heel and toe marker. As illustrated in Figure 8.2, the heel strike was determined by the first observed lowest position of the heel marker and toe off by the latest lowest position of the toe marker.

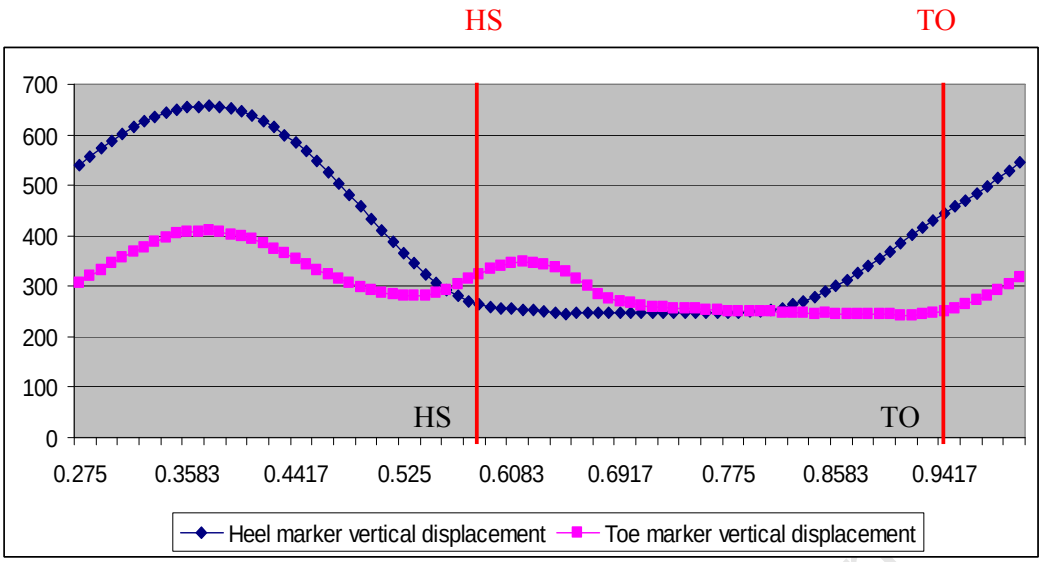

Figure 8.2. Typical vertical displacement -time graph of the heel and toe markers of a treadmill trial.

Abbreviations: *HS = heel strike, TO = toe off.*

Kinematic and EMG data were collected simultaneously using the Oxford Metrics Vicon System (Oxford Metrics, Oxford, England). The kinematic and EMG data analysis process has already been described fully in Chapter 3 *Research Methodology*, section 3.3 Data Analysis.

8.2.3 Statistical Analysis

Trials for each participant were not averaged as there was only one trial for each time interval. Data were compared in three different ways: over time; injured vs. uninjured at each time interval; and pain condition injured group vs. uninjured at the 6 min interval.

8.2.3.1 Data analysis over time

Data were analysed over time for runners in the injured and in the uninjured groups. Data were analysed separately using a One–Way Repeated Measure ANOVA. A significant main effect was analysed further using a Tukey *post hoc* analysis test. For the injured runners, data were compared at 30 s, 3 min, 6 min and at the time point

coinciding with the onset of pain. The reason for not using the final 9 min time point was that most injured runners developed pain before that time resulting in a large decrease in the number of participants that were measured at 9 min (6 min = 17 participants; 9 min = 9 participants).

8.2.3.2 Data analysis for injured vs. uninjured groups at each time interval

Data were analysed and compared between the injured participants before they felt pain and the matched uninjured participants in each time interval. For reasons already explained in section 8.2.3.1 data were processed at 30 s, 3 min and 6 min time points between the two groups. Differences between the two groups were compared using the t test for independent variables.

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8.2.3.3. Data analysis for pain development in the injured group vs. the uninjured group at the 6 min interval

In the third analysis, the data of the runners with Achilles tendinopathy and the uninjured runners were compared at 6 min. This time point (6 min) was selected because 57% of the runners with Achilles tendinopathy developed pain before 9 mins (Table 8.2). Data were analysed using an independent t- test.

The false discovery procedure was used to account for multiple comparisons (Curran-Everett, 2000). Statistical significance was accepted as $p \leq 0.05$ before the adjustments were made. To measure the magnitude of a treatment effect, the effect size was reported when statistical differences were observed. The Eta squared (r^2) method was applied to measure effect size when the ANOVA test was performed (e.g. over time) (Tabachnick and Fidell, 1989), and the standard method of Cohen (d) was used to measure the effect size when an independent t-test was performed (e.g.: injured vs. uninjured at each time interval and pain condition injured vs. uninjured at 6 min) (Cohen, 1988).

8.3 Results

The running velocity during each test was similar between the two groups: injured runners: 2.72 ± 0.62 m/s; uninjured runners: 2.71 ± 0.38 m/s, p = 0.968.

8.3.1. Over time

8.3.1.1 Temporal distance and kinematics

There were no significant changes in the temporal distance and kinematic parameters of runners in the uninjured group over time (30 s and 3, 6 and 9 min) (Table 8.3 and 8.4). Similarly there were no significant changes in the temporal distances and kinematic parameters of runners in the injured group over time (30s, 3min, 6min and pain) (Table 8.5 and 8.6).

Table 8.3. Temporal distance parameters of runners in the uninjured group over time (Values are depicted as mean ± SD).

	UNINJURED (N=34)				
	30s	**3min**	**6min**	**9min**	**p value**
Stride Ln (m)	1.39 ± 0.15	1.39 ± 0.13	1.37 ± 0.16	1.34 ± 0.33	0.775
Stride F (strides/s)	85.1 ± 5.4	84.5 ± 6.3	85.0 ± 5.1	85.2 ± 5.2	0.956
CT (s)	0.35 ± 0.03	0.35 ± 0.03	0.34 ± 0.04	0.34 ± 0.03	0.510
VSDsp (cm)	8.9 ± 1.4	8.8 ± 1.5	9.4 ± 1.7	9.2 ± 1.4	0.309

Abbreviations: *CT = contact time; VSDsp = vertical sacral displacement.*

Table 8.4. Kinematics variables of runners in the uninjured group over time (Values are depicted as mean ± SD).

	UNINJURED (N=34)				
	30s	**3min**	**6min**	**9min**	**p value**
Hic (°)	43.9 ± 9.3	42.3 ± 8.6	44. ± 11.8	41.5 ± 7.8	0.633
Hto (°)	2.8 ± 7.8	1.6 ± 8.6	1.9 ± 12.7	0.4 ± 7.4	0.817
HROM (°)	41.2 ± 7.3	40.8 ± 6.3	42.5 ± 5.9	41.1 ± 5.2	0.724
KswE (°)	17.2 ± 7.2	17.0 ± 7.7	15.5 ± 11.5	16.4 ± 8.1	0.867
Kic (°)	24.3 ± 9.0	22.6 ± 7.5	20.5 ± 11.8	21.7 ± 7.8	0.432
Kst (°)	40.3 ± 6.4	40.9 ± 7.4	40.0 ± 7.6	39.9 ± 7.6	0.960
KROM (°)	16.0 ± 6.1	18.3 ± 5.8	19.2 ± 7.2	18.7 ± 4.8	0.172
Asw (°)	-10.2 ± 6.1	-10.0 ± 6.5	-8.9 ± 6.7	-7.9 ±6.1	0.514
Aic (°)	-9.3 ± 5.8	-9.7 ± 5.7	-8.6 ± 6.5	-8.6 ± 4.8	0.834
Ast (°)	-23.3 ± 3.0	-23.5 ± 3.4	-22.8 ± 3.4	-22.7 ± 3.4	0.780

Abbreviations: *Hic = hip angle at initial contact; Hto = hip angle at toe off; HROM = hip range of motion; KswE = knee angle at terminal swing phase; Kic = knee angle at initial contact; Kst = knee angle at stance phase; KROM = knee range of motion; Asw = ankle angle at swing phase; Aic = ankle angle at initial contact; Ast = ankle angle at stance phase.*

Table 8.5. Temporal distance parameters of injured runners over time (Values are depicted as mean ± SD).

	INJURED (N=21)				
	30s	**3min**	**6min**	**Pain**	**p value**
Stride Ln (m)	1.41 ± 0.17	1.42 ± 0.17	1.41 ± 0.23	1.39 ± 0.19	0.983
Stride F (strides/s)	96.2 ± 55.3	84.2 ± 6.0	83.8 ± 4.9	83.8 ± 4.9	0.412
CT (s)	0.35 ± 0.04	0.34 ± 0.03	0.34 ± 0.04	0.35 ± 0.07	0.664
VSDsp (cm)	9.1 ± 2.0	8.8 ± 1.5	9.3 ± 2.0	9.2 ± 2.1	0.847

Abbreviations: *CT = contact time; VSDsp = vertical sacral displacement.*

Table 8.6. Kinematic variables of injured runners over time (Values are depicted as mean ± SD).

	INJURED (N=21)				
	30s	**3min**	**6min**	**Pain**	**p value**
Hic (°)	44.7 ± 11.2	48.4 ± 12.3	47.9 ± 14.1	45.8 ± 12.0	0.789
Hto (°)	5.3 ± 9.9	3.2 ± 11.2	5.8 ± 11.6	4.0 ± 9.7	0.894
HROM (°)	37.6 ± 15.5	43.2 ± 9.4	42.1 ± 14.7	41.9 ± 9.2	0.564
KswE (°)	18.4 ± 6.0	21.7 ± 7.4	22.2 ± 8.7	18.4 ± 7.1	0.298
Kic (°)	24.3 ± 7.9	25.5 ± 9.2	26.3 ± 7.3	23.5 ± 7.3	0.750
Kst (°)	38.9 ± 5.5	40.6 ± 4.9	40.9 ± 5.9	40.1 ± 5.5	0.765
KROM (°)	14.1 ± 6.4	15.2 ± 5.7	14.6 ± 7.2	16.6 ± 5.5	0.690
Asw (°)	-10.6 ± 4.8	-7.1 ± 7.0	-9.5 ± 4.1	-8.7 ± 4.9	0.288
Aic (°)	-9.9 ± 4.2	-7.4 ± 6.5	-8.7 ± 4.7	-7.0 ± 5.3	0.314
Ast (°)	-22.5 ± 5.4	-22.7 ± 2.3	-22.9 ± 3.3	-21.9 ± 3.0	0.886

Abbreviations: *Hic = hip angle at initial contact; Hto = hip angle at toe off; HROM = hip range of motion; KswE = knee angle at terminal swing phase; Kic = knee angle at initial contact; Kst = knee angle at stance phase; KROM = knee range of motion; Asw = ankle angle at swing phase; Aic = ankle angle at initial contact; Ast = ankle angle at stance phase.*

8.3.1.2 Integrated electromyography (IEMG)

Integrated IEMG activity pre (100 ms before initial contact), post (100 ms after initial contact), during the whole gait cycle and stance phase was similar over time for uninjured (Table 8.7 and 8.8) and injured runners (Table 8.9 and 8.10).

Table 8.7. IEMG activity (%*s) Pre (100 ms before initial contact), Post (100 ms after initial contact) of uninjured runners over time (Values are depicted as mean ± SD).

	UNINJURED (N=34)				
	30s	**3min**	**6min**	**9min**	**p value**
TApre	16.0 ± 6.3	17.9 ± 7.2	18.7 ± 6.5	19.4 ± 5.0	0.219
TApost	18.8 ± 10.9	16.8 ± 5.7	17.7 ± 6.6	17.2 ± 4.6	0.743
BFpre	22.2 ± 12.1	22.2 ± 8.0	24.7 ± 9.1	22.5 ± 9.5	0.741
BFpost	14.0 ± 5.9	14.7 ± 4.6	15.2 ± 6.3	16.8 ± 7.6	0.359
PEpre	9.0 ± 9.2	7.5 ± 4.5	7.5 ± 3.0	8.5 ± 6.5	0.747
PEpost	19.7 ± 11.4	18.1 ± 5.9	25.1 ± 15.2	20.7 ± 10.3	0.117
GMpre	10.4 ± 6.8	9.6 ± 4.5	10.3 ± 5.4	8.9 ± 5.4	0.767
GMpost	21.1 ± 10.5	22.7 ± 10.9	22.7 ± 9.8	24.2 ± 12.6	0.817
LGpre	7.4 ± 5.7	10.0 ± 8.3	7.2 ± 5.8	9.0 ± 6.9	0.337
LGpost	17.8 ± 10.5	19.7 ± 9.8	19.9 ± 8.6	21.3 ± 10.7	0.610
RFpre	9.6 ± 9.0	10.0 ± 6.6	10.5 ± 8.1	7.9 ± 4.3	0.541
RFpost	24.2 ± 13.3	23.7 ± 9.6	27.5 ± 10.9	22.8 ± 9.6	0.402

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

Table 8.8. IEMG activity of the whole gait cycle (GC) and in the stance phase (SP) (%*s) of uninjured runners over time (Values are depicted as mean ± SD).

	UNINJURED (N=34)				
	30s	**3min**	**6min**	**9min**	**p value**
TA GC	13012 ± 1085	12405 ± 1544	12390 ± 1730	12469 ± 1561	0.310
TA SP	5998 ± 1580	5257 ± 1241	5260 ± 1908	4955 ± 1338	0.062
BF GC	13081 ± 2256	12645 ± 1347	13101 ± 2359	12772 ± 3249	0.833
BF SP	6418 ± 1555	6538 ± 1603	6642 ±2359	6746 ± 3090	0.945
PE GC	13127 ± 2149	12573 ± 1501	13085 ± 2020	13112 ± 1622	0.596
PE SP	9297 ± 2262	8746 ± 2385	9868 ± 2863	9973 ± 1966	0.186
GM GC	12608 ± 1040	11801 ± 1403	12973 ± 1352	12977 ± 2826	0.102
GM SP	7467 ± 1864	7040 ± 1920	7669 ± 2008	8252 ± 2780	0.357
LG GC	12842 ± 2048	12731 ± 1720	12569 ± 1908	13225 ± 2197	0.638
LG SP	9645 ± 2868	9024 ± 2356	9540 ± 2729	10107 ± 2471	0.476
RF GC	13083 ± 3046	12782 ± 1813	13488 ± 3534	12750 ± 3143	0.752
RF SP	8916 ± 2599	8428 ± 2104	8801 ± 2301	8407 ± 1753	0.743

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

Table 8.9. IEMG activity (%*s) Pre (100 ms before initial contact), Post (100 ms after initial contact) of injured runners over time (Values are depicted as mean ± SD).

	INJURED (N=21)				
	30s	**3min**	**6min**	**Pain**	**p value**
TApre	14.7 ± 4.9	14.8 ± 5.4	14.2 ± 4.4	12.8 ± 5.4	0.614
TApost	16.7 ± 8.6	15.1 ± 6.8	14.9 ± 8.9	16.7 ± 7.2	0.859
BFpre	20.3 ± 12.6	21.0 ± 13.5	19.6 ± 11.7	20.9 ± 14.4	0.989
BFpost	17.0 ± 7.2	17.1 ± 7.4	15.7 ± 6.1	19.6 ± 9.0	0.483
PEpre	8.4 ± 6.2	8.2 ± 6.8	10.2 ± 8.6	9.7 ± 9.2	0.833
PEpost	17.2 ± 9.5	20.5 ± 15.0	21.7 ± 9.8	17.6 ± 8.7	0.526
GMpre	14.9 ± 5.6	11.7 ± 7.8	11.7 ± 6.4	12.3 ± 8.3	0.732
GMpost	21.9 ± 12.2	19.3 ± 10.1	18.8 ± 6.8	20.2 ± 9.5	0.905
LGpre	8.1 ± 6.3	7.1 ± 3.2	8.2 ± 4.9	9.5 ± 9.9	0.780
LGpost	17.1 ± 11.1	18.5 ± 13.0	20.4 ± 13.4	18.8 ± 11.1	0.890
RFpre	11.7 ± 7.4	11.8 ± 6.8	12.4 ± 7.3	8.5 ± 5.6	0.285
RFpost	21.2 ± 8.7	20.9 ± 11.0	22.6 ± 11.2	18.2 ± 8.2	0.588

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

Table 8.10. IEMG activity of the whole gait cycle (GC) and in the stance phase (SP) (%*s) for injured runners over time (Values are depicted as mean ± SD).

	INJURED (N =21)				
	30s	**3min**	**6min**	**Pain**	**p value**
TA GC	12463 ± 1595	13375 ± 3754	11825 ± 2093	12524 ± 2905	0.436
TA SP	6037 ± 1978	6326 ± 2725	5674 ± 2593	6167 ± 3109	0.907
BF GC	12965 ± 2010	12978 ± 1384	12621 ± 1079	13233 ± 1633	0.732
BF SP	6798 ± 2118	7048 ± 2216	6749 ± 2233	7376 ± 863	0.849
PE GC	12653 ± 1176	12975 ± 2021	13462 ± 1817	12947 ± 2569	0.671
PE SP	8917 ± 2419	9618 ± 3458	8982 ± 1756	9106 ± 3414	0.887
GM GC	12793 ± 2998	12100 ± 1336	11751 ± 1351	12339 ± 2114	0.682
GM SP	6776 ± 1750	7012 ± 1769	6816 ± 1881	6893 ± 2141	0.990
LG GC	13129 ± 1907	12846 ± 1212	12306 ± 1471	12638 ± 1559	0.487
LG SP	9559 ± 3567	9409 ± 3108	8767 ± 2696	9197 ± 3427	0.908
RF GC	12826 ± 1756	12661 ± 1251	12712 ± 1792	11852 ± 1725	0.277
RF SP	7661 ± 2089	7481 ± 1990	7277 ± 2022	7715 ± 2719	0.943

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

8.3.1.3 Peak EMG and time to peak EMG during the stance phase

Runners in the uninjured and injured groups did not have any significant changes in the peak EMG values and time to peak EMG during the stance phase of running over time (Table 8.11 and 8.12 respectively).

Table 8.11. Peak EMG activity in the stance phase (P SP) (%) and time of occurrence of peak EMG activity in the stance phase (T SP) (% gait cycle) of uninjured runners over time (Values are depicted as mean ± SD).

	UNINJURED (N=34)				
	30s	**3min**	**6min**	**9min**	**p value**
TA PSP (%)	337 ± 130	285 ± 95	312 ± 108	301 ± 97	0.295
TA TSP (s)	54± 14	52 ± 14	49 ± 13	49 ± 11	0.431
BF PSP (%)	307 ± 115	314 ± 122	335 ± 224	312 ± 169	0.170
BF TSP (s)	58 ± 14	63 ± 14	57 ± 13	54 ± 13	0.070
PE PSP (%)	449 ± 131	422 ± 125	474 ± 235	495 ± 109	0.327
PE TSP (s)	61 ± 9	62 ± 10	62 ± 6	62 ± 9	0.895
GM PSP (%)	353 ± 137	340 ± 131	382 ± 150	399 ± 166	0.576
GM TSP (s)	58 ± 9	56 ± 7	58 ± 6	59 ± 6	0.767
LG PSP (%)	423 ± 145	413 ± 105	409 ± 122	460 ± 131	0.437
LG TSP (s)	67 ± 11	65 ± 11	62 ± 6	64 ± 7	0.248
RF PSP (%)	470 ± 237	440 ± 162	439 ± 231	419 ± 118	0.788
RF TSP (s)	63 ± 10	63 ± 11	58 ± 6	61 ± 8	0.093

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

Table 8.12. Peak EMG activity in the stance phase (P SP) (%) and time of occurrence of peak EMG activity in the stance phase (T SP) (% gait cycle) of injured runners over time (Values are depicted as mean ± SD).

	INJURED (N=21)				
	30s	**3min**	**6min**	**Pain**	**p value**
TA PSP (%)	275 ± 133	283 ± 115	237 ± 102	276 ± 110	0.650
TA TSP (s)	55 ± 18	64 ± 22	52 ± 14	59 ± 19	0.269
BF PSP (%)	327 ± 132	330 ± 125	306 ± 135	362 ± 137	0.661
BF TSP (s)	60 ± 15	62 ± 15	58 ± 14	59 ± 15	0.909
PE PSP (%)	402 ± 150	453 ± 222	384 ± 96	431 ± 178	0.644
PE TSP (s)	63 ± 9	62 ± 10	62 ± 11	64 ± 12	0.927
GM PSP (%)	309 ± 111	320 ± 109	303 ± 82	348 ± 222	0.871
GM TSP (s)	59 ± 11	59 ± 9	59 ± 11	60 ± 11	0.989
LG PSP (%)	440 ± 211	448 ± 171	398 ± 175	435 ± 203	0.890
LG TSP (s)	69 ± 11	64 ± 7	61 ± 12	66 ± 11	0.156
RF PSP (%)	381 ± 138	370 ± 134	378 ± 119	377 ± 125	0.996
RF TSP (s)	55 ± 9	57 ± 8	60 ± 14	59 ± 10	0.487

Abbreviations: *TA = tibialis anterior, BF = biceps femoris, PE = peroneus longus, GM = gluteus medius, LG = lateral gastrocnemius, RF = rectus femoris.*

8.3.2. Injured vs. uninjured groups at each time interval

8.3.2.1. Temporal Distance parameters and Kinematics parameters

There were no significant differences in temporal distance parameters and kinematic parameters at different time intervals time between runners in uninjured and injured groups.

8.3.2.2. Integrated electromyography (IEMG)

Although it was not significant after correction, the IEMG of the gluteus medius muscle during the running cycle tended ($p = 0.026$) to be lower in injured runners compared with uninjured runners at the 6 min interval of the running trial (Figure 8.3). Similarly, the IEMG of the rectus femoris muscle tended to be lower during the stance phase (significant before correction, $p = 0.036$) in the injured group compared with the uninjured group at the same time interval (Figure 8.4). There were no other significant differences in IEMG activity during the whole cycle or in the stance phase between the two groups for the other muscles at the different time points. Furthermore, there were no differences in IEMG activity 100 ms before or after heel strike between both groups at similar time points.

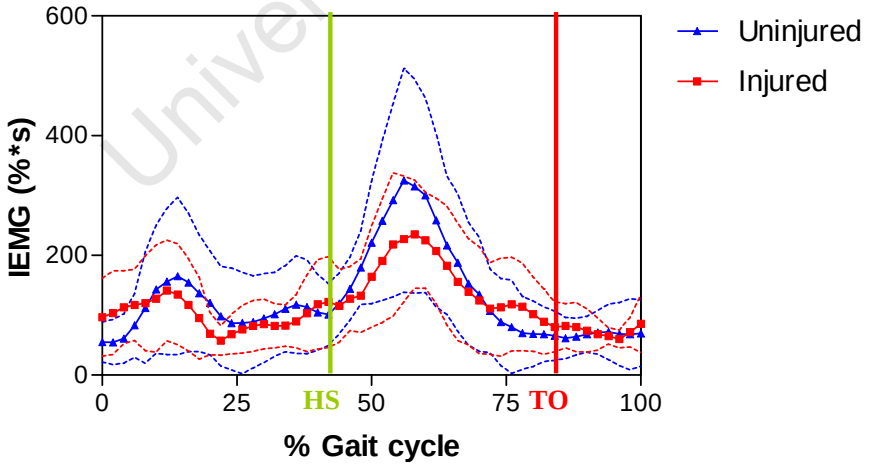


Figure 8.3. EMG activity of gluteus medius at 6 min in the uninjured vs. injured runners. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

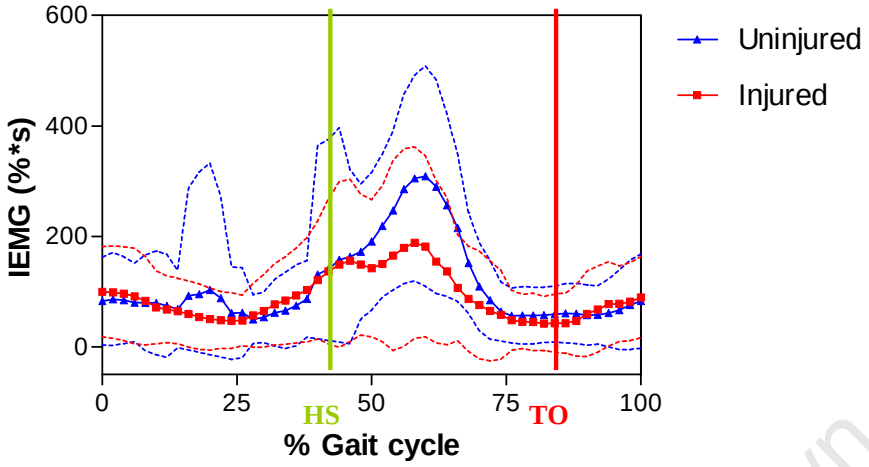


Figure 8.4. EMG of the rectus femoris muscle at 6 min in the uninjured vs. injured runners. Values are presented throughout the gait cycle (0-100%). And variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

8.3.2.3. Peak EMG and time to peak EMG during the stance phase

The peak EMG activity of the tibialis anterior muscle tended to be higher (significant before correction, p=0.027) for runners in the uninjured group compared with the injured group during the stance phase at the 6 min time interval (Figure 8.5).

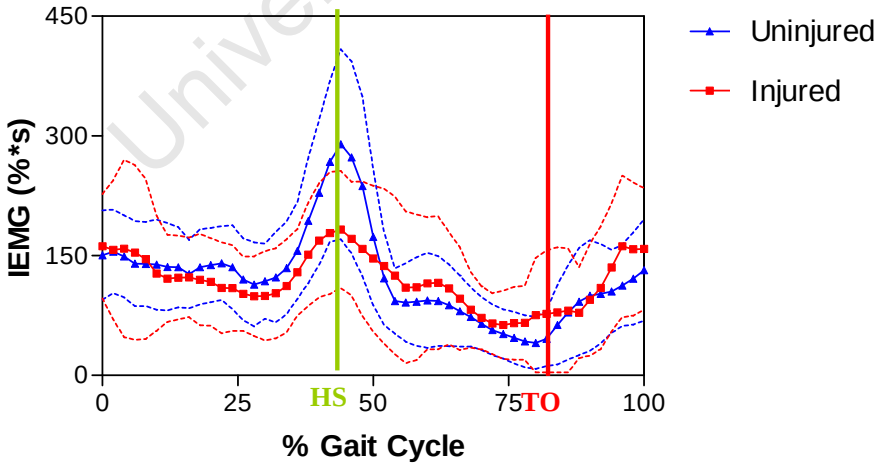
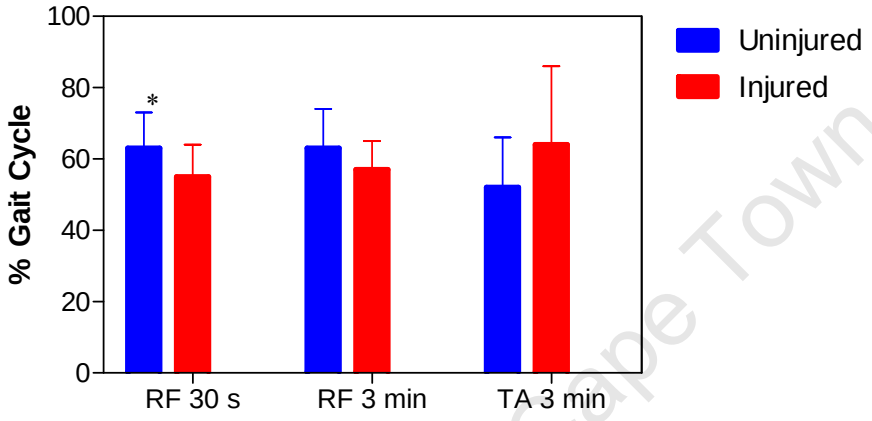


Figure 8.5. EMG tibialis anterior activity at 6 min in the uninjured vs. injured runners. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

The peak EMG activity of the rectus femoris muscle occurred significantly earlier in the injured group at 30 seconds and tended to occur earlier at the 3 min interval (significant before correction, p = 0.046) when compared with the runners in the uninjured group. On the other hand, peak EMG activity of the tibialis anterior muscle at the 3 min time interval tended to occur later in the injured group compared to the uninjured group (significant before correction, p = 0.026) (Figure 8.6).



* p = 0.006

Effect size RF 30 s *d* = 0.84 i.e. large effect size

Figure 8.6. Time of peak EMG during stance phase

Abbreviations: *RF = rectus femoris, TA = tibialis anterior.* .

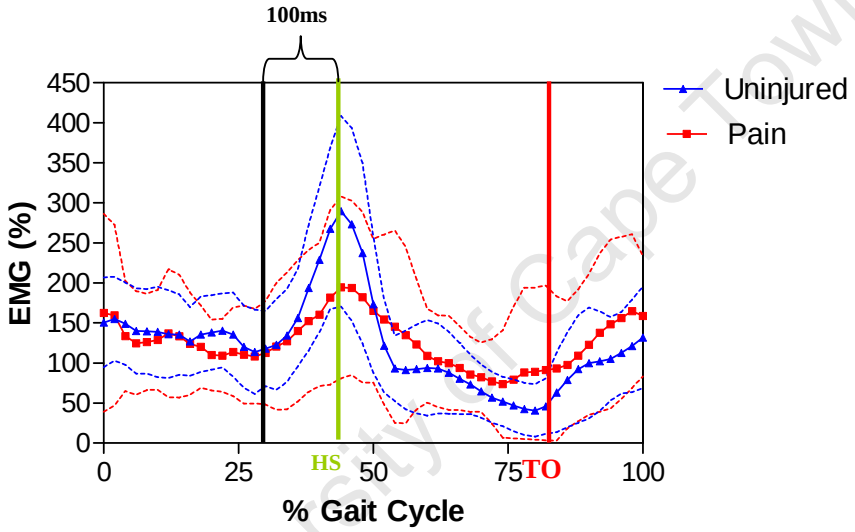
8.3.3. Pain development in the injured group vs. uninjured group at 6 min interval

8.3.3.1 Temporal distance and kinematics

There were no statistical differences in temporal distance parameters and kinematic parameters between injured runners when they felt pain and the uninjured runners at the 6 min time interval.

8.3.3.2 Integrated electromyography (IEMG)

There were no significant differences in the IEMG activity of the lower limb muscles when data were analysed for the whole gait cycle or during the stance phase. However, there were some statistical differences when data were analysed 100 ms before or after heel strike. Tibialis anterior IEMG activity at 100 ms before heel strike was significantly lower in the injured runners compared with the uninjured runners (Figure 8.7). Similarly, IEMG activity of the rectus femoris at 100 ms after heel strike which was also significantly lower in the injured compared with the uninjured runners (Figure 8.8).

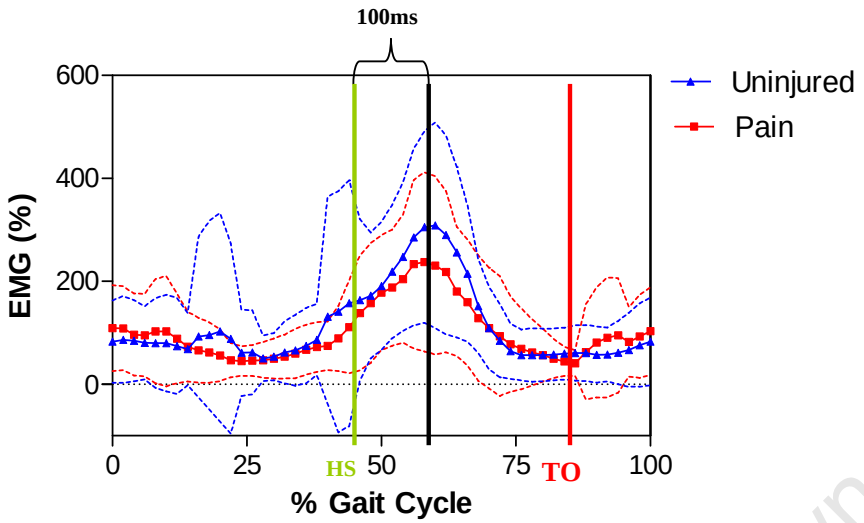


* IEMG 100 ms before HS significantly different between uninjured at 6 min and pain condition injured runners p = 0.029.

Effect size TA pre *d* = 0.985 i.e. large effect size

Figure 8.7. EMG of the tibialis anterior muscle in the uninjured group at 6 min vs. the pain condition in the injured runners. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.

Abbreviations: *HS = heel strike, TO = toe off.*



* IEMG 100 ms after HS significantly different between uninjured at 6 min and pain condition injured runners p = 0.026

Effect size RF post *d* = 0.956 i.e. large effect size

Figure 8.8. EMG rectus femoris muscle activity in the uninjured group at 6 min vs. the pain condition in the injured runners. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation
Abbreviations: *HS = heel strike, TO = toe off.*

8.3.3.3 Peak EMG and time to peak EMG during stance phase

Peak EMG activity was not significantly different between the two conditions. However, for the tibialis anterior muscle, the time to reach peak EMG activity tended to be delayed (significant before correction, p = 0.039) in the pain condition for the injured runners when compared to the uninjured runners at the 6 min interval (Figure 8.9).

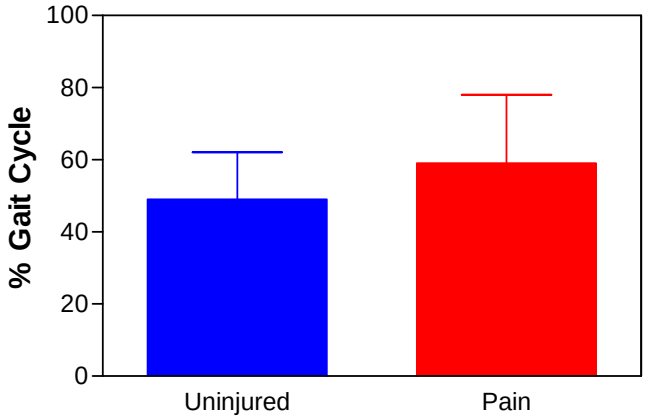


Figure 8.9. The time of peak EMG activity in the tibialis anterior muscle during the stance phase.

8.4 Discussion

The main findings of this study were that runners with Achilles Tendinopathy did not show any changes in kinematic and EMG activity during treadmill running before or during the onset of pain. However, when compared with a control group of uninjured runners, runners with Achilles tendinopathy had reduced EMG activity of the tibialis anterior and rectus femoris muscles when pain developed during running.

The mechanism for the pain experienced by patients with Achilles tendinopathy is not clear and is still being investigated (Khan et al., 2000). Recently it has been suggested that the presence of glutamate (Alfredson et al., 1999) and ionotrophic glutamate receptor *N*-methyl-D-aspartate (NMDA) in the Achilles tendon might be associated with the pain experienced by runners who suffer from Achilles tendinopathy (Alfredson et al., 2001).

Glutamate is an important pain modulator in the central nervous system (Alfredson and Lorentzon, 2002). A very recent study (Scott et al., 2008), showed that tenocytes may be involved in the control of extracelluar glutamate levels. It is possible that the increase in glutamate may impact nociception (unconscious sensation of pain).

It has been shown that EMG activity increased in different jaw and neck muscles when glutamate was evoked to promote pain in the masseter muscle at rest, but similar results were not found when the head was set in a different position (Svensson et al., 2004). The effect of an increase in glutamate concentration on EMG activity of lower limb muscles in runners with Achilles tendinopathy has not yet been investigated and was not measured in this study. However, in the present study, neither the EMG activity nor the kinematics changed in the injured runners when they felt pain during running (Tables 8.5, 8.6, 8.9, 8.10 and 8.12). The reasons for this are not clear but may be related to methodological issues. Firstly biomechanical parameters were measured after a very short period following the onset of pain (less than 30 seconds after the pain developed). It is possible that runners with a chronic injury such as Achilles tendinopathy may require a longer exposure to painful stimuli before kinematic and EMG adjustments are made in response to pain. Secondly, these runners had low injury grades (I or II), which meant that they normally felt pain only after running or during running but the pain does not restrict their run. Therefore, the maintenance of the same biomechanical pattern when the runners felt pain may be explained the lower level of pain felt by the runners. It is suggested that further studies are conducted where longer exposure to more intense pain is assessed.

Similar to the injured runners, the uninjured runners did not alter their biomechanics during the 9 minute run (Tables 8.3, 8.4, 8.7, 8.8 and 8.11). This result was expected. Although EMG activity and kinematics can change during fatigue conditions (Derrick et al., 2002; Weist et al., 2004), the runners in this study were only required to run at a comfortable self-selected running speed for a short time and were not likely to develop fatigue.

The kinematics and muscle activity between injured and uninjured runners at different time intervals before pain developed during running was also studied. The results in Figures 8.3, 8.4 and 8.5 show that EMG activity tended to be different between the two groups at the 6 minutes interval. The IEMG activity of the rectus femoris in the injured group tended to be reduced during the stance phase (Figure 8.4). These results are similar to that already reported and discussed in Chapter 5. Study 2 *Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*, when runners were tested during 10 running trials

on a 10 m pathway. These data support the hypothesis that a weakness of the quadriceps eccentric action during early stance phase may be associated with Achilles injury. Similarly, the peak EMG activity of the rectus femoris muscle occurred earlier in the stance phase in the injured runners compared with the uninjured runners at 30 s and at the 3 mins time interval (Figure 8.6). This response was not observed at 6 mins, but there was a tendency to decrease IEMG activity of this muscle in the stance phase (Figure 8.4), showing that after a period of 3 minutes when EMG activity may be more synchronized between the two groups but muscle activity was reduced in the injured runners. In contrast to these findings, it has been shown that runners with patellofemoral syndrome had similar EMG activity of the vastus lateralis, vastus medialis and rectus femoris during the stride cycle (running at an intensity of 80% of the training pace, at 12km/h) (MacIntyre and Robertson, 1992). Therefore, runners with different injuries might show different muscle activity patterns during running.

The observation that there was a tendency for a reduction in the IEMG activity of the gluteus medius at 6 min (Figure 8.3) was also supported by the results that were already presented in Chapter 5 Study 2 *Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*. The only difference between the observations from these two studies is that the reduction of EMG activity was noticed only 100 ms after heel strike in the previous study (Chapter 5) while the reduction occurred during the whole gait cycle in this study. The possible consequence of a reduced IEMG activity of GM in stance phase is that it may indicate a weak gluteus medius. Therefore, it can be suggested that the weakness in this muscle may result in an increase in femoral adduction and knee and tibial rotation, and this may increase pronation. It should however be noted that pronation was not measured in this study.

The peak IEMG activity of the tibialis anterior muscle tended to be lower in injured runners during the stance phase at the 6 min time interval (Figure 8.5). Furthermore, peak EMG activity of the tibialis anterior muscle tended to occur later in the injured runners compared with the uninjured runners at the 3min time interval (Figure 8.6). In this thesis it was reported in Chapter 5 Study 2 *Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*, that pre EMG activity (100 ms before heel strike) of the tibilais anterior was

significantly decreased in the injured group. Although similar results for the same time period (100ms before heel strike) of the running cycle were not found in this study, the pattern of reduced EMG activity of this muscle was maintained in this study. Furthermore, as is discussed later when runners felt pain, there was a reduction of IEMG at 100 ms before heel strike, which is similar to the results that were already presented and discussed in Chapter 5 Study 2 *Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*. The tendency of the reduced peak EMG activity of tibialis anterior during the stance phase during the 6 min interval and the delay in peak EMG activity at 3 min may indicate a reduction in the control of ankle during the early stance phase when this muscle acts eccentrically (McClay et al., 1990). Although, similar sagittal plane ankle kinematics were found between in the two groups in this study frontal and transverse plane kinematics were not measured in this study. It is therefore suggested that further studies should investigate these other planes of movement.

Finally, in this study, EMG activity during pain in the injured group was compared with the data collected in the uninjured group at the 6 min time interval. In this analysis, kinematic parameters were not affected in the pain condition when compared with uninjured runners. Previous studies when runners with iliotibial band syndrome (ITBS) were subjected to a 20 mins run at a pace that caused exhaustion, an increase in knee flexion angle at heel strike, maximum foot inversion and knee internal rotation velocity were observed in these runners, compared with controls (Miller et al., 2007). Therefore, it can be speculated that perhaps the running speed and duration selected in this study were not sufficient to result in biomechanical alterations. Although further studies need to examine the effect of fatigue on runners with Achilles tendinopathy, there is an ethical concern about this type of study because there the risks that the protocol may exacerbate the injury.

In contrast muscle activity 100 ms before and after heel strike was significantly different between the two groups. Runners in the injured group had significantly lower tibialis anterior IEMG activity 100 ms before heel strike (Figure 8.7) and also a delay in peak EMG activity of this muscle during the stance phase (Figure 8.9). This first result is similar to the findings which were already reported in Chapter 5 Study2

Tibialis anterior activity during the late swing phase is responsible for the control of plantarflexion and the initiation of dorsiflexion (Cavanagh, 1990). The reduced IEMG activity of the TA in the injured group may indicate that the increase in ankle stiffness is as a result of the injury and this may affect the dorsiflexion of the foot. Although a significant difference in ankle dorsiflexion was not found, Figure 8.10 illustrates that injured runners tended to have a reduced ankle dorsiflexion during the stance phase when they felt pain, compared with controls. It has previously been suggested that the gastrocnemius and soleus weakness in runners with Achilles tendinopathy may restrain dorsiflexion during at the beginning of the support phase of running (Hess et al., 1989). However, according to the results of the present study, the restraint in dorsiflexion and motion control appears to be related to the reduced tibialis anterior activity and not necessarily the gastrocnemius as previously suggested.

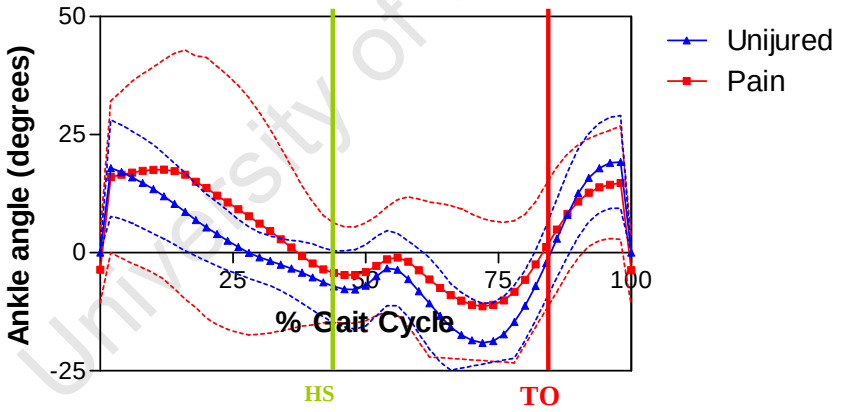


Figure 8.10. Ankle sagittal plane kinematics. Values are presented throughout the gait cycle (0-100%) and variance is displayed as standard deviation.
Abbreviations: *HS = heel strike, TO = toe off.*

The reduction in IEMG activity of the rectus femoris muscle at 100 s after heel strike is also a finding that is similar to the findings that were previously observed and discussed in Chapter 5 Study 2 *Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*. The reduced IEMG activity of the rectus femoris muscle during this period may affect the

role of this muscle during the early stance phase to restrain the movement of the tibia and to absorb impact during the stance phase (Novacheck, 1998b).

EMG has been used as an important diagnosis tool for patients with lower back pain (Nouwen and Bush, 1984; Linsinski, 2000). Patients with lower back pain show a delay in reaction time measured by the EMG activity (Panagiotacopulos et al., 1998) and increase in EMG activity due to muscle tension (Nouwen and Solinger, 1979). Although in the present study, the muscle activity at the start of pain was not altered in runners with Achilles tendinopathy, the results of this study do indicate that EMG activity can be different in injured runners before or during pain development when compared with controls. Furthermore, although several muscles were investigated, tibialis anterior and rectus femoris were the muscles, which are frequently associated with an altered pattern response. Therefore, the practical application from the result of this study is that rehabilitative exercises that stimulate the muscle activity of these muscles may be beneficial in the prevention and treatment of this condition.

CHAPTER 9. SUMMARY AND CONCLUSION

The overall purpose of this thesis was concerned with the multi-factorial aetiology of Achilles tendinopathy in particular intrinsic risk factors that may be associated with Achilles tendinopathy. The first specific aim of the thesis was to determine if any of the following three groups of intrinsic risk factors were associated with Achilles tendinopathy in runners: 1) training and injury history of runners, anthropometric, flexibility and lower limb alignment; 2) biomechanical parameters (kinetic, kinematics and muscle activity); 3) variability of biomechanical parameters (kinetic, kinematic and muscle activity) from stride-to-stride and between participants in injured and uninjured groups. The second specific aim of the thesis was to determine if changes in sensory input (by changing shoe hardness, and allowing pain to develop during running) alter biomechanical parameters (kinetic, kinematic and muscle activity). In order to address these two specific aims the related literature was critically reviewed using evidence-based approach. Thereafter, a series of research studies were undertaken to systematically address the aims of the thesis, in order to ultimately add to the body of knowledge about the risk factors for this injury condition. A case-control study design was used in which thirty four uninjured runners and twenty-one runners with Achilles tendinopathy were recruited and a series of measurements including training and injury history, anthropometrical measurements and biomechanics parameters (kinetic, kinematic and muscle activity) parameters were recorded.

The first main finding of the thesis reveal that runners with Achilles Tendinopathy have a higher prevalence of previous running injury, had run for more years, have reduced flexibility, and are heavier and taller than uninjured runners. The second main finding was that runners with Achilles tendinopathy had a reduced IEMG activity of the tibialis anterior muscle 100 ms before heel strike and of the rectus femoris muscle during the stance phase of running. Knee range of motion (KROM) from initial contact to mid-stance also tended to be less in the runners with Achilles tendinopathy, but vertical and anterior-posterior forces were similar between the two groups. These results therefore showed that although both groups experienced similar impact forces but runners with Achilles tendinopathy had reduced muscle activity and lower

KROM, which indicate that the injured runners are less able to attenuate the impact that travels through the leg with each stride.

The third main finding of the thesis was that stride-to-stride movement variability was similar in runners with Achilles tendinopathy and uninjured runners, indicating that both groups of runners adjust their biomechanics to the external conditions with the same variability from stride to stride. However, a novel finding was that there was a reduced inter-participant variability for kinetic and muscle activity parameters in runners with Achilles tendinopathy. This finding suggests that uninjured runners but not runners with Achilles tendinopathy adjust their biomechanics according to their different lower limb and foot morphology, functional behaviour or external and internal sensory signals but this requires further investigation.

In summary, the main findings of these first three studies can now be added to the existing body of knowledge on risk factors for Achilles tendinopathy in runners. In Tables 9.1 and 9.2, the results were incorporated to the findings of the current literature on intrinsic and extrinsic risk factors in Achilles tendinopathy, using the evidence base medicine (EBM) criteria introduced in Chapter 2. *A Review of the Literature* (Obremskey et al., 2005). These tables illustrate how this thesis (highlighted in the table as Azevedo et al., 2008) has contributed to enhance the knowledge on the understanding of the risk factors of Achilles tendinopathy in runners.

Table 9.1. Extrinsic risk factors associated to Achilles tendinopathy using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Training errors	**D. Increase training distance**	
	Positive association:	
	Retrospective study: (Clement et al., 1984)	II
	Case control: (McCrory et al., 1999)	III
	E. Increase training intensity	
	Positive association:	
	Retrospective study: (Clement et al., 1984)	II
	Case control: (McCrory et al., 1999)	III
	F. Limited warm-up	
	Positive association:	
	Prospective study: (Milgrom et al., 2003)	I
	Expert opinion: (Barr and Harrast, 2005)	V
	No association:	
	Case control: (Azevedo et al., 2008)	III
Lack of Stretching	**No association:**	
	Case control: (Azevedo et al., 2008)	III
Cross-training	**No association:**	
	Case control: (Azevedo et al., 2008)	III
Footwear	**Positive association:**	
	Retrospective study: (Clement et al., 1984)	II
	Systematic review of level II: (Smart et al., 1980)	II
	Expert opinion: (McKenzie et al., 1985)	V
	No association:	
	Case control: (Azevedo et al., 2008)	III
Running surface	**Negative association:**	
	Case control: (McCrory et al., 1999)	III
	No association:	
	Case control: (Azevedo et al., 2008)	III

Table 9.2. Intrinsic risk factors associated to Achilles tendinopathy using the evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Age	**Positive association:**	
	Prospective study: (Kannus et al., 1989)	I
	Retrospective study: (Clement et al., 1984)	II
	Systematic review level I-III studies: (Alfredson and Lorentzon, 2000)	III
Gender	**Positive association (Male):**	
	Retrospective study: (Clement et al., 1984)	II
	Case control: (Astrom and Rausing, 1995)	III
Increase height, body weight, % body fat	**A. Increased Height:**	
	Positive association:	III
	Case control: (Azevedo et al., 2008)	
	B. Increased Weight:	
	Positive association:	
	Case control: (Azevedo et al., 2008)	III
	B. Increased % body fat:	
	No association:	
	Positive association:	
	Case control: (Azevedo et al., 2008)	III
Less running experience	**Negative association:**	
	Case control: (Azevedo et al., 2008)	III
History of previous injury	**Positive association:**	
	Case control: (Azevedo et al., 2008)	III
Muscle weakness	**A. Muscle weakness**	
	Positive association	
	Prospective study: (Alfredson et al., 1998b)	I
	Case control: (McCrory et al., 1999)	III
	Expert opinion: (Paavola et al., 2002)	V
Inflexibility	**Positive association**	
	Retrospective study (Clement et al., 1984)	II
	Systematic review level I-III studies: (Hess et al., 1989; Kvist, 1994; Alfredson and Lorentzon, 2000)	III

Table 9.2 continued. Intrinsic risk factors associated to Achilles tendinopathy using evidence-based medicine (EBM) criteria.

Risk factor	Study information	Level of Evidence (I-V)
Lower limb abnormalities	**Positive association**	
	Prospective study: (Lun et al., 2004)	I
	Retrospective study: (Clement et al., 1984)	II
	Case study: (McCrory et al., 1999)	
	Systematic review level I-III studies: (Kvist, 1994)	III
	No association:	
	Case control: (Azevedo et al., 2008)	III
Biomechanical factors	**A. Functional overpronation**	
	Positive association:	
	Retrospective study: (Clement et al., 1984; Kvist, 1994)	II
	Systematic review of level II	II
	(Jones, 1998; Paavola et al., 2002)	
	Systematic review level I-III studies: (Kvist, 1994)	III
	Case series: (Clancy, Jr. et al., 1976)	IV
	B. Dynamic overpronation	
	Positive association:	
	Case control: (McCrory et al., 1999)	III
	C. Sagittal plane kinematics parameters	
	No association:	
	Case control: (Azevedo et al., 2008)	III
	C. Kinetic parameters	
	No association:	
	Case control: (McCrory et al., 1999)*Azevedo et al., 2008)*	III
	G. Muscle activity parameters	
	Positive association:	
	Case control: (Azevedo et al., 2008)	III
	H. Running biomechanics variability	
	Positive association:	
	Case control: (Azevedo et al., 2008)	III
Genetic predisposition	**Positive association:**	
	Case control: (Mokone et al., 2006; September et al., 2008)	III

The fourth main finding of this study was that no specific biomechanical adaptations in runners with or without Achilles tendinopathy could be detected in response to different shoe conditions (soft vs. hard). However, muscle activity of the rectus femoris was consistently reduced in the Achilles tendinopathy group compared with the uninjured runners, and this was independent of the shoe condition (hard or soft). This finding indicates that shoes with different shock absorbing proprieties do not alter the reduced muscle activity observed previously in another chapter (Chapter 5, Study 2 *Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners*) in runners with Achilles tendinopathy.

The final main finding of this thesis was that runners with Achilles Tendinopathy did not show any evidence of altered biomechanics or muscle activity pattern at the onset of pain. However, runners with Achilles tendinopathy did show a steady reduced muscle activity of the tibialis anterior and rectus femoris muscle before and in response to the development of pain during running.

A limitation of this thesis is that it is not definitively conclusive whether the findings reported in the specific studies were an adaptative response to injury or an aetiological causative factor. Therefore, similar studies to those reported in this thesis should be reproduced using a prospective design and/or analysing the biomechanical response with rehabilitation intervention.

The originality of this thesis lies in the investigation of muscle activity in an Achilles running injured population as previous studies have only analysed kinetic and kinematic parameters. Another innovative feature of the thesis was the investigation of different sensory inputs (shoe hardness and pain) on kinetic, kinematic and muscle activity parameters in a running injured population. The practical application of the findings is that runners with Achilles tendinopathy may require specific rehabilitation exercises that stimulate the muscle activity of rectus femoris and tibialis anterior.

It is recommended that further prospective studies or randomized controlled intervention studies would be necessary to determine a cause-effect relationship between kinetic, kinematic and muscle activity factors and Achilles tendon injury.

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APPENDICES

Appendix 1

Evidence- based medicine criteria

Copied from Journal of Bone and Joint Surgery (Obremskey et al., 2005)

Levels of Evidence for Primary Research Question[1]

	Types of Studies			
	Therapeutic Studies—Investigating the Results of Treatment	Prognostic Studies—Investigating the Effect of a Patient Characteristic on the Outcome of Disease	Diagnostic Studies—Investigating a Diagnostic Test	Economic and Decision Analyses—Developing an Economic or Decision Model
Level I	· High-quality randomized controlled trial with statistically significant difference or no statistically significant difference but narrow confidence intervals · Systematic review[2] of Level-I randomized controlled trials (and study results were homogeneous[3])	· High-quality prospective study[4] (all patients were enrolled at the same point in their disease with ≥80% follow-up of enrolled patients) · Systematic review[2] of Level-I studies	· Testing of previously developed diagnostic criteria in series of consecutive patients (with universally applied reference "gold" standard) · Systematic review[2] of Level-I studies	· Sensible costs and alternatives; values obtained from many studies; multiway sensitivity analyses · Systematic review[2] of Level-I studies
Level II	· Lesser-quality randomized controlled trial (e.g., <80% follow-up, no blinding, or improper randomization) · Prospective[4] comparative study[5] · Systematic review[2] of Level-II studies or Level-I studies with inconsistent results	· Retrospective[6] study · Untreated controls from a randomized controlled trial · Lesser-quality prospective study (e.g., patients enrolled at different points in their disease or <80% follow-up) · Systematic review[2] of Level-II studies	· Development of diagnostic criteria on basis of consecutive patients (with universally applied reference "gold" standard) · Systematic review[2] of Level-II studies	· Sensible costs and alternatives; values obtained from limited studies; multiway sensitivity analyses · Systematic review[2] of Level-II studies
Level III	· Case-control study[7] · Retrospective[6] comparative study[5] · Systematic review[2] of Level-III studies	· Case-control study[7]	· Study of nonconsecutive patients (without consistently applied reference "gold" standard) · Systematic review[2] of Level-III studies	· Analyses based on limited alternatives and costs; poor estimates · Systematic review[2] of Level-III studies
Level IV	Case series[8]	Case series	· Case-control study · Poor reference standard	· No sensitivity analyses
Level V	Expert opinion	Expert opinion	Expert opinion	Expert opinion

1. A complete assessment of the quality of individual studies requires critical appraisal of all aspects of the study design.
2. A combination of results from two or more prior studies.
3. Studies provided consistent results.
4. Study was started before the first patient enrolled.
5. Patients treated one way (e.g., with cemented hip arthroplasty) compared with patients treated another way (e.g., with cementless hip arthroplasty) at the same institution.
6. Study was started after the first patient enrolled.
7. Patients identified for the study on the basis of their outcome (e.g., failed total hip arthroplasty), called "cases," are compared with those who did not have the outcome (e.g., had a successful total hip arthroplasty), called "controls."
8. Patients treated one way with no comparison group of patients treated another way.

This chart was adapted from material published by the Centre for Evidence-Based Medicine, Oxford, UK. For more information, please see www.cebm.net.

Fig. E-1

Level-of-evidence rating system used by JBJS-A. (Reprinted from: Instructions to Authors, *The Journal of Bone and Joint Surgery*.)

Appendix 2

UNIVERSITY OF CAPE TOWN

Department of Human Biology

Faculty of Health Sciences
University of Cape Town
Observatory Cape 7925
South Africa
Tel: + 27 21 406 6235
Fax: + 27 21 448 7226

CLINICAL DIAGNOSIS OF ACHILLES TENDINOPATHY

Name:_____ Date: _____

Code:_____

Referred from: _____

Clinical criteria	**Present**
Gradual progressive pain over the posterior lower leg- Achilles tendon area.	
Early morning pain	
Early morning stiffness	
History of swelling over the Achilles tendon area	
Tenderness to palpation over the Achilles tendon	
Palpable nodular thickening over the affected Achilles	
Positive "shift" test (movement of the nodular area with plantar-dorsiflexion)	

Signature: _____

References:

1. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athlete. AM. J. Sports Med 2002; 30: 287-305
2. Kader D, Saxena A, Movin T, Maffulli N. Achilles tendinopathy: some aspects of basic science and clinical management. Br J Sports Med 2002; 36:239-49/

Appendix 3

Participant information document

Biomechanical and muscle recruitment of different running injuries.

Dear Runner

Thank you for your participation in this study. The aim of this study is to analyze the biomechanical and muscle activity characteristics of runners with: Achilles tendinosis and anterior knee pain syndrome and compare them with injury-free runners.

The study also aims to evaluate the biomechanics and muscle recruitment alterations with the use of different shoes, during the development of pain symptoms and after healing.

Gait Analyses

You will be asked to fill out a comprehensive questionnaire detailing your personal, medical and training history.

At the Gait Analyses Laboratory you will undergo to some anthropometric measurements. We will place some electrodes and reflective markers on your skin. After that, you will be invited to do a series of running trials barefoot and with two different shoes at a self-selected running speed. You will then be asked to run on a treadmill at a self selected running speed until the first symptom of injury discomfort appears: at which time you will only have to run 30 more seconds. On the third and final test you will run again at the lab with shoes. If you are an injury-free runner on these two final tests you will just have to run on the treadmill for 10 to 15 minutes and then run at the lab both at a self selected running speed.

For the injured runners, we would like to re-analyze your biomechanics when you become injury-free to further evaluate any changes that may have occurred after healing. Thus, we would appreciate if you could contact us when it happens.

If you have any questions, don't fail to ask. We look forward to your participation.

Yours faithfully,

Liane Beretta de Azevedo

Appendix 4

Informed Consent

Biomechanics Study

I, ……, have been fully informed of the nature of this study and hereby give consent to act as a participant for the research.

I am fully aware of the procedures involved at the gait analysis running trials in the Gait Analysis Laboratory.

I am aware that I may withdraw my consent and participation in the research project at any time.

I consent that the data collected may be used for scientific purposes and publications in a confidential manner.

I understand the implications of my informed consent and any question I may have had have been answered to my satisfaction.

	Name	Signature	Date
Participant	…….	……	……
Researcher	…….....	……	……
Witness	…….	……	…….

Appendix 5

UNIVERSITY OF CAPE TOWN

Research Ethics Committee
E46 Room 26, Old Main Building Groote
Schuur Hospital, Observatory, 7925
Queries : Xolile Fula
Tel : (021) 406-6492 Fax: 406-6411
E-mail : Xfula@curie.uct.ac.za

10 October 2003

REC REF: 287/2003 (288/2003 - Protocol correct number)

A/Prof. MP. Schwellnus
Human Biology
Sport Science Institute

Dear Prof. Schwellnus

THE RELATIONSHIP BETWEEN BIOMECHANICS AND NEUROMUSCULAR CHARACTERISTICS IN COMMON RUNNING INJURIES

Thank you for submitting your study to the Research Ethics Committee for review.

Date Considered: 03 October 2003

Decision: Approved
The following comments are made

- Please include the insurance clause in the consent form.
- Since this is a proposal for a higher degree, greater care needs to be taken regarding spelling and grammatical errors, which should have been corrected during proof reading.

Attached please find the list of members who attended the meeting

Please quote the REC REF number in all your correspondence

Yours sincerely

PROF T.ZABOW
CHAIRPERSON

10/10/2003

Hi Prof Schwellnus
Your reference no is
actually 288/2003 &
not 287/2003. In
future correspondence
please quote 288/20
Thank you
[illegible]

Appendix 6

Runner's Questionnaire

For office use:

Date: ___/___/___ **1.** | | | |

Referred from: **2.** |

- ☐ SSISA (1)
- ☐ UCT Practice (2)
- ☐ Running Club (3)
- ☐ Others (4)
- ☐ Non injured runner (5)

A. Personal Details:

1.Name: _____ **3.** | | |

2. Birth date: _____ **Age:** _____ **4.** | |

3. Gender: Male: ☐ (1) Female: ☐ (2) **5.** |

4. Postal address: _____

_____

5. Phone number: _____

6. E-mail address: _____

7. Occupation: _____

8. Current running club: _____

9. Dominant leg: ☐Left (1) ☐Right (2) **6.** |

B. Medical History

1. Do you have one of the following medical conditions? **7.** | | | |

- ☐ Neurological disorders (1)
- ☐ Diabetes mellitus (2)
- ☐ Physical deformities of the lower limb (3)
- ☐ Surgery on the knee region (4)
- ☐ Surgery on the foot region (5)
- ☐ Surgery on the ankle region (6)

2. Do you take any medication? **8.** |

- ☐ No (1)
- ☐ Yes(2) Please indicate name and dosage per day

Medication	Dosage

C. Injury History:
If you are an uninjured runner go to question 7

For office use:

1.Time between injury and this visit: _____ days **9.** ☐☐☐

2.What type of injury do you have? **10.** ☐

☐ Acute (sudden injury) (1)

☐ Chronic(progressively worse over a long period of time)(2)

3.How would you grade your injury ? **11.** ☐

☐ Grade I: pain only AFTER running. (1)

☐ Grade II: pain DURING running does not restricting running. (2)

☐ Grade III: pain DURING running, restricting running. (3)

☐ Grade IV: restricting running. (4)

4.Where is the site of your injury? **12.** ☐

☐ Lower back (1)

☐ Hip (2)

☐ Pelvis (3)

☐ Right thigh (4)

☐ Left thigh (5)

☐ Right knee (6)

☐ Left knee (7)

☐ Right ankle (8)

☐ Left ankle (9)

☐ Right Foot (10)

☐ Left foot (11)

5.What was the diagnosis of your injury? **13.** ☐

☐ Patellofemoral pain syndrome (1)

☐ Medial tibial stress syndrome (2)

☐ Achilles Tendinosis (3)

☐ Iliotibial band friction syndrome (4)

☐ Plantar fasciitis (5)

☐ Others (6) Specify: _____

For office use:

6. Which of the following can you relate to the cause of the injury? **14.** ☐☐☐☐

- ☐ Change in running shoes (1)
- ☐ Running on hard surface (2)
- ☐ Running on soft surface (3)
- ☐ Increased hill running (4)
- ☐ Increased in training volume (4)
- ☐ Increase on training intensity (5)
- ☐ Don't know (6)
- ☐ Others (7) Specify: _____

7.Have you ever suffered from a previous injury? **15.** ☐

- ☐ Yes (1)
- ☐ No (if no skip to section C-Training History) (2)

8.What was diagnosis of your other injury? **16.** ☐

- ☐ Patellofemoral pain syndrome (1)
- ☐ Medial tibial stress syndrome (2)
- ☐ Achilles Tendinosis (3)
- ☐ Iliotibial band friction syndrome (4)
- ☐ Plantar fasciitis (5)
- ☐ Others (6) Specify: _____

9.When did this injury occur? **17..** ☐☐☐
_____ **months ago**

D. Training History:

1.For how many years have you run regularly? **18.** ☐☐
_____years

2.Level of competition: **19.** ☐

- ☐ Elite (1)
- ☐ Competitive club level (2)
- ☐ Recreational competitive (3)
- ☐ Recreational only (4)

For office use:

3.Write here the approximate percentage of time per week you run on the following surfaces

Surface	% time training		
Road		**20.**	
Track		**21.**	
Trail/off road		**22.**	
Grass		**23.**	
Sand		**24.**	

4.How many pairs of running shoes do you usually train with? **25.** ☐☐
_____ pairs of running shoes

5.For how many kilometers on average do you run in each pair of running shoes? **26.** ☐☐☐☐

_____ km

6.Do you use orthotics: **27.** ☐

☐ Yes (1)

☐ No (2) If no go to question 9 below).

7.What type of orthotics? **28.** ☐

☐ Soft commercial orthotics (not specifically made for you) (1)
If you use soft orhtotics goes to question 9.

☐ Custom orthotics (made for you) (2)

8.Where are your orhtotics build up: **29.** ☐

☐ Heel rise (1)

☐ Heel rise on inside (2)

☐ Heel rise on outside (3)

☐ Arch support (4)

☐ Support over big toe (5)

☐ Support over small toe.(6)

9. Do you do any cross training? **30.** ☐

☐ Yes (1)

☐ No (2) (If no go to question 11 on next page)

For office use:

10. What type of cross training do you do? How often?

Cross training	Frequency per week
Swimming (1)	
Cycling (2)	
Weight training (3)	
Field sports (cricket, rugby, soccer…)(4)	
Others(5). Specify:	

31. ☐

32. ☐

11. Do you stretch? **33.** ☐

☐ Yes (1)

☐ No (2) (If no go to question 13 below)

12. How often and for how long do you stretch? **34.** ☐

_____ days per week

_____ minutes per day **35.** ☐

13. Do you warm up before the running session? **36.** ☐

☐ Yes (1)

☐ No (2) (If no skip to section D on next page)

14. How do you warm up? **37.** ☐

☐ Walking (1)

☐ Running slower (2)

☐ Stretching (3)

☐ Others (4) Specify: _____

15. For how long do you warm up before the running session? **38.** ☐

_____ min

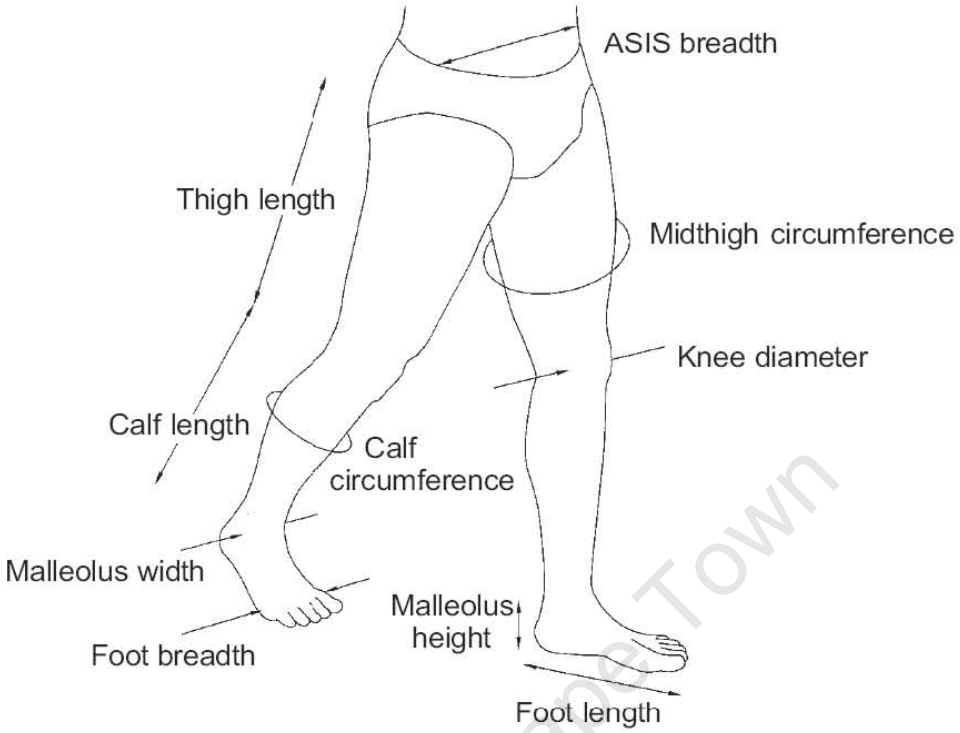
1. Please write in this training diary, all information that you can remember from your training in the last 5 years:

Occasion	Weekly distance	Average speed min/km	Training sessions per week	Training sessions consider hard per week	Hill training per week	Longest run of the week
0-4 weeks						
1 month ago						
2 months ago						
3 months ago						
4 months ago						
5 months ago						
6 months ago						

Appendix 7

Anthropometrical assessments for biomechanical model

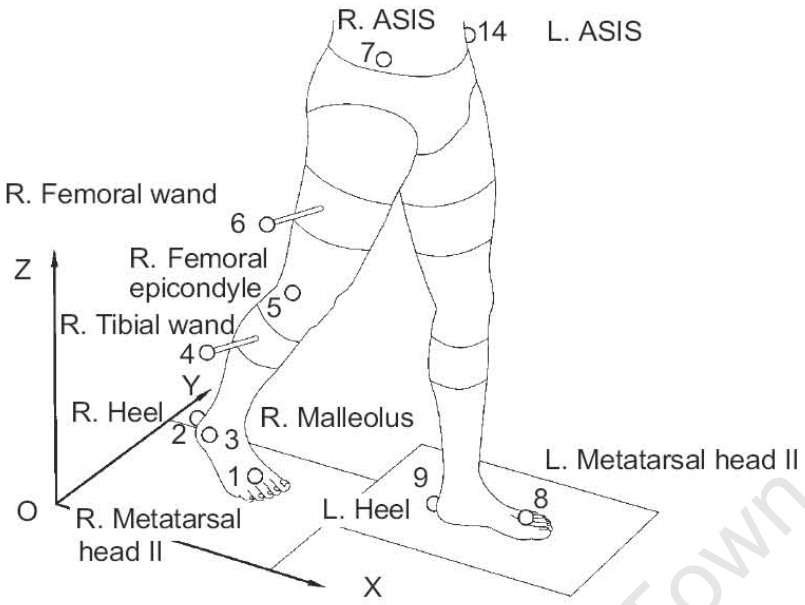
Parameter description	Measurement (mm)
Total body height (m)	Use the tape at the wall of the treadmill lab. The heel, buttocks and upper back should be touching the vertical upright. The chin should be level and not lifted.
Total body mass (kg)	Scales in the treadmill room: measure the mass of the participant without all clothes (except the short) removed.
ASIS breadth (mm)	Caliper- measure the horizontal distance between the anterior superior iliac spines.
R&L thigh length (mm)	Tape– the vertical distance between the superior point of the greater trochanter of the femur and the superior margin of the lateral tibia is measured.
R&L mid-thigh circumference (mm)	Tape – the circumference is measured perpendicular to the long axis of the leg at a point midway between the trochanteric and tibial landmarks.
R&L calf length (mm)	Tape/caliper – the vertical distance between the superior margin of the lateral tibia and the lateral malleolus
R&L mid-calf circumference (mm)	Tape – is measured perpendicular to the long axis of the lower leg, at the maximum circumference.
R&L knee diameter (mm)	Calipers – maximum diameter of the knee across the femoral epicondyles
R&L foot length (mm)	Calipers – the distance from the posterior margin of the heel to the tip of the longest toe
R&L foot breadth (mm)	Calipers – the breadth across the distal ends of the metatarsal I and V is measured
R&L malleolus height (mm)	Calipers – with the participant standing, the vertical distance from the standing surface to the lateral malleolus
R&L malleolus width (mm)	Calipers – the maximum distance between the medial and lateral malleoli



Appendix 8

Modified Helen Hayes Marker set (Vaughan et al., 1999)

Marker placement	Description marker position
Sacrum	Placed on the skin mid-way between the posterior superior iliac spines
Anterior Superior Iliac Spine (L & R)	Placed directly over the left or right anterior superior iliac spine
Femoral wand (L & R)	A 4 inch wand is placed on the left leg over the lower lateral 1/3 surface of the thigh.
Lateral Femoral epicondyle (L & R):	Placed on the lateral epicondyle of the left or right knee
Tibial wand (L & R)	Similar to the thigh markers, these are placed over the lower 1/3 of the shank to determine the alignment of the ankle flexion axis.
Lateral malleolus (L & R)	Placed on the lateral malleolus along an imaginary line that passes through the transmalleolar axis
Left Metatarsal Head (L &R)	Placed over the second metatarsal head, on the mid-foot side of the equinus break between fore-foot and mid-foot
Calcaneous (L & R)	Placed on the calcaneus at the same height above the plantar surface of the foot as the toe marker



Appendix 9

EMG data excluded from different chapters

A. Data excluded in Chapter 5 Study 2: Lower Limb Kinetic, Kinematic and Muscle Activity Variables as Risk Factors Associated with Achilles Tendinopathy in Runners (soft shoe) and Chapter 7. Study 4: The Effect of Different Shoe Hardness on Lower Limb, Kinetic, Kinematic and Muscle Activity Variables in Runners with Achilles Tendinopathy (soft and hard shoes).

	SOFT		HARD	
	Uninjured	Injured	Uninjured	Injured
TA	0	0	0	0
BF	3	1	1	1
PERO	4	1	3	1
GM	3	2	3	2
LGAS	2	1	3	1
RF	3	1	2	1

B. Data excluded in Chapter 8. Study 5: The Effect of Pain Development during Running on Lower Limb Kinetic, Kinematic and Muscle Activity Variables in Runners with Achilles Tendinopathy.

Injured participants

	30s	3min	6min	9min	Pain
TA	1	1	0	0	0
BF	0	0	0	0	0
PERO	1	1	0	0	0
GM	6	5	4	3	5
LGAS	1	1	1	1	0
RF	1	1	0	1	1

Uninjured participants

	30s	3min	6min	9min
TA	3	1	7	3
BF	1	1	6	4
PERO	2	4	6	5
GM	11	12	13	14
LGAS	2	3	7	5
RF	1	2	6	5

Appendix 10

Pilot Study: EMG normalization for gait analysis

Introduction:

The role of electromyogram (EMG) normalization is to compare EMG activities between different or the same muscles on different days and individuals (Lehman and McGill, 1999). There are different methods used for EMG normalization, including: normalization against an isometric maximal voluntary contraction (MVC), peak dynamic method and mean dynamic method (Burden et al., 2003).

The isometric MVC is the only method which represents the degree of muscle activity required during gait. In this method, each data point of the gait EMG is divided by the peak EMG from an isometric MVC (Dubo et al., 1976). In the peak dynamic method, each point of the processed EMG is divided by the peak value recorded from the same EMG (Burden et al., 2003). Finally, the mean dynamic method divide each data point by the mean value recorded from the same EMG (Yang and Winter, 1984).

The decision about which is the best normalisation method is based on the reduction of intra-participant variability (or stride-to-stride variability) and inter-participant variability (variability between participants). Previous studies have shown that the mean dynamic method and the peak dynamic method reduce inter-participant variability in comparison with submaximal MVC normalization and un-normalized EMG during walking (Yang and Winter, 1984). Furthermore, others studies have shown that mean dynamic method reduces inter-participant variability if compared to peak dynamic method (Shiavi et al., 1987).

Although MVC and submaximal MVC have been used to normalize isotonic tasks and walking gait (Burden and Bartlett, 1999; Benoit et al., 2003), the use of an isometric contraction to normalize a non-isometric contraction such as walking or running has been questioned (Dubo et al., 1976). Recently a study suggested the use of isokinetic MVC for gait normalisation (Burden et al., 2003). The authors found that this method is less reliable than other methods of EMG normalization as it produces a higher inter-and intra- participant variability (Burden et al., 2003). However, other

methods such as walking at a controlled speed have never been tested as a normalization EMG method for running gait.

Therefore, the aim of this study was to compare inter-and intra-participant variability between mean dynamic, peak dynamic, mean walk normalisation and un-normalized EMG methods during a running gait.

Methods:

Ten (N=10) injury-free participants (7 females and 3 males) were recruited from the University population to participate in the study (age: 28.6 ± 7.2 years, body mass: 61.8 ± 9.9 kg, height: 1.69 ± 0.09 m).

Electromyogram (EMG) signals were recorded through a Telemyo Noraxon EMG System (Noraxon USA, Inc) at a sampling rate of 2000 Hz. EMG signals were detected from each muscle by two surface triode electrodes (Thought Technology Triode MIEP01-00, Montreal, Canada). Electrodes were placed on the right leg over the visual midpoint of the contracted muscle belly of the muscles: tibialis anterior (TA), lateral gastrocnemius (LG), rectus femoris (RF) and biceps femoris (BF) and a single electrode (ground electrode) was placed over the patella. The cables and amplifiers were taped to the skin to minimize movement artefacts.

EMG activity from isometric maximal voluntary contraction (MVC) from the knee flexors and extensors and ankle plantar and dorsiflexion muscle groups were recorded using a BIODEX dynamometer (Biodex Medical Systems, New York).

After the isometric test, participants were directed to the Gait Analysis Laboratory where they performed 3 walking trials in barefoot condition at a speed controlled by a metronome (56 beats per minute) (Vic Firth VM-500, Boston, MA, USA). To identify the heel strike and toe–off, a footswitch (2-FSR Sensor Foot Contact Lead, Noraxon USA, Inc) was taped under the heel and toe of each participant. The participants then performed 7 running trials at a self selected running speed with their own running shoes.

Data Analysis

Data analysis was processed for five valid running trials and for one walking trial. EMG data were filtered by a notch filter of 50 Hz and then by a bandpass filter with a cut-off frequency between 15 and 500 Hz. root mean square (RMS) amplitudes of all EMG signals were calculated over consecutive periods of 50 ms.

The EMG signal from one stride length was temporarily normalized to 51 data points for the walking and running trials (0 to 100% gait cycle, intervals of 2%). The peak and average RMS of the EMG data for each gait cycle were divided by each data point of the EMG signal. The mean EMG for a walk cycle and the mean EMG from the highest 1000 ms MVC contraction were used as a denominator for the walk and MVC normalisation respectively. Values were expressed as a percentage of the mean RMS EMG (%). For the EMG data not normalised, EMG filtering and RMS amplitude was calculated and the value was expressed in microvolts (uV).

The intra-participant variability (or stride-to-stride variability), and inter-participant variability (variability between participants) were calculated for each individual over 5 strides (1 stride per trial).

The following equation was applied:

$$VR = \frac{\sum_{i=1}^{k}\sum_{j=1}^{n}(X_{ij}-\bar{X}_i)^2/k(n-1)}{\sum_{i=1}^{k}\sum_{j=1}^{n}(X_{ij}-\bar{X})^2/(kn-1)}$$

where k is the number of time intervals over the gait cycle (i.e. 51 data points), n is the number of trials in this case five (intra-participant variability), or the number of participants (n=10) (inter-participant variability). X_{ij} is the EMG value at the ith interval for the jth trial (intra-participant variability) or participant (inter-participant variability). $\overline{X_i}$ is the mean of EMG value at the i^{th} interval over the j gait cycle (intra—participant variability) or participant (inter-participant variability). Finally, X is the

mean of EMG, kinetic or kinematic values, i.e.: $X = 1/k \sum Xi$. These equations were described by Burden et al., (2003).

Independent t-tests were used to compare intra-participant variability. Statistical significance was accepted as $p \leq 0.05$.

Results

The results in Table 1 show that the intraparticipant variance ratios for TA and RF were significantly higher for the peak dynamic method than the mean dynamic method and walk methods of normalisation. Furthermore, the variance ratio values for LG normalised with the peak dynamic method was significantly higher than MVC and walk methods. Finally, the variance ratio values for BF normalized with the peak dynamic method were significantly higher than mean dynamic method and un-normalised EMG.

Table 1: Intra-participant variability measured by variance ratio of un-normalized and normalized using the mean dynamic, peak dynamic, isometric MVC and mean walk methods during running (N=10).

	TA	LG	RF	BF
Mean dynamic	0.53 ± 0.27	0.27±0.22	0.22±0.17	0.17±0.26
Peak dynamic	0.72±0.25*	0.47±0.23†	0.50±0.19*	0.69±0.21††
MVC	0.56±0.31	0.31±0.09	0.09±0.23	0.23±0.27
Walk	0.53±0.29	0.29±0.18	0.18±0.19	0.19±0.27
Un-normalized	0.58±0.25	0.25±0.20	0.20±0.23	0.23±0.27

* Variance ratio from peak dynamic method significantly greater ($P<0.05$) than mean dynamic method and walk methods.

† Variance ratio from peak dynamic method significantly greater ($P<0.05$) than MVC and walk methods

†† Variance ratio from peak dynamic method significantly greater ($P<0.05$) than mean dynamic method and un-normalized.

Inter–participant variability tends to be lower with the mean dynamic method than other methods or un-normalized EMG.

Table 2: Inter-participant variability of data not normalised and normalised using the mean dynamic, peak dynamic, isometric MVC and mean walk methods.

	TA	LG	RF	BF
Mean dynamic	0.71	0.32	0.21	0.66
Peak dynamic	0.93	0.57	0.42	0.85
MVC	0.95	0.42	0.50	0.87
Walk	0.96	0.63	0.46	0.77
Un-normalized	0.88	0.60	0.37	0.74

Discussion:

The main results of this study were that the peak dynamic method provides higher intra-participant variability than other methods. Furthermore, inter-participant variability is lower for the mean dynamic method. Therefore, according to these results, the peak dynamic method can be considered the least reliable while the mean dynamic method is the most reliable as it presents a lower inter-and intra-participant variability.

The findings of this study support other studies which found that the mean dynamic method reduces inter-participant variability when compared to the peak dynamic method (Shiavi et al., 1987) or un-normalized EMG (Yang and Winter, 1984). However, some authors suggest that the reduction in inter-participant variability may remove true biological variability between participants (Burden et al., 2003). In our study the variance ratio for the mean dynamic method was in the range of 0.21 to 0.71, which can be considered slightly higher than the normal standards expected from reliability studies (variance ratio < 0.30) (Kadaba et al., 1985; Bogey et al., 2003). These results indicate that although the mean dynamic method presents a lower

variance ratio than other methods, it still shows some variance between participants, which may indicate their true biological variability.

Although, one study found that the use of MVC as a normalization method promotes lower intra-participant variability than mean or peak dynamic method (Knutson et al., 1994); in this study MVC tended to produce higher variance ratio for the intra- and inter-participant variability than the mean dynamic methods for most of the muscles tested. Furthermore, the use of an isometric maximal voluntary contraction (MVC) normalization method has been questioned for gait analysis (Dubo et al., 1976).

The use of walk normalization seems to produce similar results to the mean dynamic method for the intra – participant variability (Table 1). However, normalization with the walk method produces one of the highest variance ratio values for the inter-participant variability (Table 2). We could perhaps speculate that the high variability between participants was affected by the fact that the walking speeds were controlled, but the running speeds were self-selected. Therefore, we suggest that further studies could be conducted at a controlled running speed to verify this hypothesis.
We therefore conclude that mean dynamic method of normalization should be applied to normalize running gait as it produces lower intra-and inter-participant variability than the other methods suggested on the literature and the walk normalization.

Appendix 11

Data with similar sample size male and female

Table 1: Participant characteristics

	Uninjured (N=21)	Injured (N=21)	p value
Age (yr)	38.9 ± 10.1	41.8 ± 9.7	0.330
Height (cm)	174.3 ± 8.0	177.8 ± 7.4	0.689
Mass (kg)	70.2 ± 10.9	77.6 ± 12.6	0.055
Years of running (yrs)	9.7 ± 7.0	13.6 ± 9.5	0.065
Training weekly distance (km)	45.7 ± 16.1	33.0 ± 18.0	0.023

Table 2: Temporal distance parameters; uninjured vs. injured

	Uninjured (N = 21)	Injured (N = 21)	p value
Speed ($m.s^{-1}$)	3.00 ± 0.41	2.97 ± 0.37	0.951
Stride length (m)	2.23 ± 0.24	2.17 ± 0.30	0.483
Stride time (s)	0.74 ± 0.04	0.74 ± 0.06	0.816
Stride frequency ($strides.min^{-1}$)	81 ± 4	82 ± 7	0.675
Contact time (s)	0.17 ± 0.01	0.17 ± 0.02	0.649

Table 3: Kinetic variables; uninjured vs. injured

	Uninjured (N = 21)	Injured (N = 21)	p value
HBF (BW)	0.21 ± 0.05	0.20 ± 0.05	0.621
HPF (BW)	0.15 ± 0.02	0.16 ± 0.04	0.172
VIF (BW)	1.34 ± 0.20	1.45 ± 0.23	0.140
VLR (BW/s)	42.87 ± 9.31	44.79 ± 11.27	0.580
VPF (BW)	2.19 ± 0.15	2.18 ± 0.23	0.875

Table 4: Kinematic variables; uninjured vs. injured

	Uninjured (N = 21)	Injured (N = 21)	p value
Hic (°)	40.7 ± 7.6	42.4 ± 7.2	0.501
Hto (°)	2.6 ± 13.0	3.8 ± 5.5	0.717
HROM (°)	35.9 ± 13.8	38.7 ± 7.1	0.455
Kic (°)	16.5 ± 6.7	20.2 ± 7.2	0.116
Kst (°)	42.8 ± 8.6	42.2 ± 4.8	0.802
KROM (°)	26.3 ± 3.9	22.0 ± 5.5	0.011*
Aic (°)	-11.4 ± 7.8	-11.5 ± 5.8	0.977
Ast (°)	-19.6 ± 11.6	-20.9 ± 3.4	0.644
Δβev (°)	21.2 ± 4.4	21.8 ± 6.1	0.151
Time Bev (s)	0.08 ± 0.02	0.07 ± 0.01	0.711

Table 5: IEMG activity (%*s) pre (100 ms before heel strike), post (100 ms after heel strike) uninjured vs. injured runners.

	Uninjured (N = 21)	Injured (N = 21)	P value
TApre	22.9 ± 5.2	17.3 ± 6.0	0.003*
TApost	11.2 ± 3.9	12.2 ± 8.0	0.592
PEpre	10.0 ± 3.5	10.2 ± 4.5	0.868
PEpost	31.2 ± 7.3	25.3 ± 10.2	0.049*
LGpre	9.3 ± 4.6	9.6 ± 5.4	0.880
LGpost	28.2 ± 9.4	23.7 ± 10.4	0.158
RFpre	14.1 ± 4.6	14.8 ± 6.5	0.679
RFpost	34.1 ± 8.2	21.6 ± 9.6	0.000*
BFpre	19.2 ± 4.7	16.1 ± 7.9	0.149
BFpost	16.8 ± 8.4	18.5 ± 7.1	0.497
GMpre	13.5 ± 3.3	13.0 ± 5.9	0.749
GMpost	25.2 ± 5.4	18.1 ± 7.9	0.004*

Table 6: IEMG activity (%*s) during stance and swing phase uninjured vs. injured runners

	Uninjured (N = 21)	Injured (N = 21)	P value
TA stance	3762 ± 922	4216 ± 1797	0.315
PE stance	9415 ± 1220	8077 ± 2447	0.045*
LG stance	9109 ± 1886	8141 ± 2579	0.192
RF stance	8124 ± 1340	6183 ± 2010	0.001*
BF stance	5971 ± 1063	6733 ± 1640	0.101
GM stance	6821 ± 923	6172 ± 1271	0.087

Appendix 12

Coefficient of variance of lower limb and functional measurements

Table 1. Lower limb anthropometrical variables of the right leg.

Right	Injured	CV	Uninjured	CV	Difference between CV
Rearfoot alignment (°)	1.6 ± 3.0	72.4	2.4 ± 1.8	185.1	112.7
Pronation angle at 45 ° (°)	5.2 ± 3.3	58.4	4.6 ± 2.7	63.0	4.6
Q angle (°)	9.7 ± 2.4	24.5	10.1 ± 2.5	24.6	0.2
Standing foot angle (°)	135.7 ± 7.5	7.7	134.8 ± 10.4	5.5	-2.2
Navicular drop (cm)	0.6 ± 0.9	33.3	0.5 ± 0.2	145.1	111.8
Forefoot alignment (cm)	2.7 ± 2.9	98.1	3.2 ± 3.1	108.8	10.7
Subtalar joint, passive range of motion (inversion) (°)	3.3 ± 2.0	75.2	2.8 ± 2.1	60.5	-14.7
Subtalar joint, passive range of motion (eversion) (°)	5.2 ± 2.6	61.9	7.1 ± 4.4	50.4	-11.5
Knee range of motion (°)	138.5 ± 7.3	5.4	140.4 ± 7.6	5.3	-0.1

Table 2. Lower limb anthropometrical values of the left leg

Left	Injured	CV	Uninjured	CV	Difference between CV
Rearfoot alignment (°)	1.6 ± 3.4	39.3	2.8 ± 2.5	35.8	-3.5
Pronation angle at 45 ° (°)	5.6 ± 3.4	90.5	6.1 ± 3.6	211.7	121.2
Q angle (°)	8.9 ± 2.0	460.4	8.7 ± 1.9	1205.9	745.5
Standing foot angle (°)	132.4 ± 9.3	22.3	134.2 ± 8.7	22.6	0.3
Navicular drop (cm)	0.7 ± 1.1	6.5	0.5 ± 0.2	7.0	0.5
Forefoot alignment (cm)	2.3 ± 1.7	30.1	2.3 ± 2.2	46.0	16.0
Subtalar joint, passive range of motion (inversion) (°)	3.1 ± 1.6	95.7	2.5 ± 1.8	75.0	-20.7
Subtalar joint, passive range of motion (eversion) (°) *	6.0 ± 3.6	72.4	8.7 ± 5.5	52.3	-20.1
Knee range of motion (°) **	135.6 ± 5.8	82.5	140.1 ± 8.1	73.5	-9.0

Table 3. Knee alignment and leg discrepancy of injured and uninjured runners.

	Injured	CV	Uninjured	CV	Difference between CV
Leg length discrepancy (cm)	1.0 ± 0.7	73.5	0.7 ± 0.6	82.5	9.0

Table 4. Body fat percentage and sit and reach results

	Injured	CV	Uninjured	CV	Difference between CV
Body fat (%)	22.1 ± 5.0	22.9	21.7 ± 5.5	25.3	2.45
Sit and reach test (cm)*	21.6 ± 10.0	46.0	27.5 ± 8.0	30.1	-16.0

Appendix 13

Outcomes of this thesis

Papers

Azevedo L.B. , Lambert M., Vaughan C.L., O'Connor C and Schwellnus M.P.– "Reduced muscle activity is associated with Achilles Tendinopathy in Runners" –**In Press** - *British Journal of Sports Medicine*.

Conference proceedings

Azevedo L.B. , Lambert M., Vaughan C.L. and Schwellnus M.P. "Biomechanics and EMG Activity During Painful Running in Runners with Achilles Tendinopathy". Abstract published on *Medicine & Science in Sports & Exercise*. 40(5) Supplement 1:S27, May 2008.

Azevedo L.B. , Lambert M., Vaughan C.L. and Schwellnus M.P. "The effect of shoe hardness on biomechanics in injured runners" – Abstract published on *Journal of Sports Sciences* December Supplement 2, Vol. 25, p3, 2007

Azevedo L.B. , O'Connor C., Lambert M., Vaughan C.L. and Schwellnus M.P. - "Biomechanics Variability in Runners with Achilles Tendinopathy."- Abstract published on *Journal of Sports Science* January 25 (3) p 236, 2006.

Azevedo L.B. , Lambert M., Vaughan C.L. and Schwellnus M.P. "Lower Limb Biomechanics and EMG Activity in Runners with Achilles Tendinopathy" - Abstract published on *Medicine & Science in Sports & Exercise*. 38(5) May Supplement:S123, 2006.

Azevedo L.B., Lambert M., Vaughan C.L., O'Connor C and Schwellnus M.P "Biomechanics Variability in Runners with Achilles Tendinopathy".- Abstract published on *Journal of Sports Science.* January 25 (3): 1-2, 2006.

Awards:

Winner of the American College of Sports Medicine Biomechanics Interest Group Student Travel Award (May, 2008) with the abstract Biomechanics and EMG Activity During Painful Running in Runners with Achilles Tendinopathy". Award of U$200.00.

Nike award winner for athletic footwear research offered by the International Society of Biomechanics (July, 2007) with the paper: "Kinetic, Kinematic and Muscle Activity Variables Associated with Achilles Tendinopathy in Runners". Azevedo L.B., Lambert M., Vaughan C.L. and Schwellnus M. Award of U$20,000.00